THE JOURNAL OF PERIODONTOLOGY

July

1959

Published by
THE AMERICAN ACADEMY OF PERIODONTOLOGY
VOLUME 30
NUMBER 3

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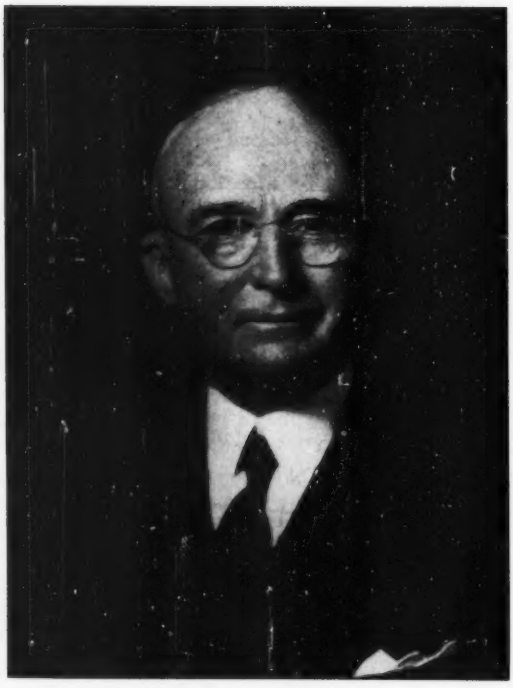
THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

THE AMERICAN ACADEMY OF PERIODONTOLOGY

1959 Annual Meeting—September 10, 11, 12, 1959

New York City, New York



ARTHUR H. MERRITT, D.D.S., M.Sc., Sc.D.

Riverdale-on-Hudson

New York, New York

This issue of the Journal of Periodontology is dedicated to Dr. Arthur H. Merritt for his innumerable contributions to dental science.

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SINCE the founding of the first national dental organization on August 3, 1859, developments of dental science have been almost spectacular by all standards. Dental health care has been improved, expanded and in more demand by the public, the understanding of dental disorders has increased, there is an ever-growing emphasis on prevention of dental disease, and the stature of the dental profession has been deservedly elevated. The growth and development of dentistry is dependent upon many, many dedicated individuals . . . it is impossible to name them all. However, one *can* site the type of men who have helped elevate dentistry to its present stature, and a good example is Dr. Arthur H. Merritt of New York.

Dr. Arthur Hastings Merritt was born in Williamsburg, Massachusetts, on May 2, 1870 and received his D.D.S. degree from New York University in 1895, and has been in the practice of dentistry since that time. He received Doctor of Science degrees from the University of Maryland, Columbia University and New York University.

Dr. Merritt has received many honors for distinguished service in the art and science of dentistry, including the Fauchard gold medal in 1932, Jarvis gold medal in 1940, Callahan gold medal in 1940, the Albert C. Fones Memorial medal in 1941, and Harry Spenadel gold medal in 1950. He is a member of many organizations and has been president of the American College of Dentists, American Dental Association and the American Academy of Periodontology.

Dr. Merritt is author of many articles and a book on "Periodontal Diseases: Diagnosis and Treatment" which has gone through three editions. Dr. Merritt has also found time to become an authority on Anglo-American historical Staffordshire as it is related to American history and has written a delightful book on the subject. He lives at 5000 Fieldstone Road, Riverdale-on-Hudson, New York.

For his long, devoted and meritorious service to dentistry, this issue of the Journal is respectfully dedicated to Dr. Arthur H. Merritt. Many of his friends have written in expressing their respect and appreciation for Dr. Merritt, and we are pleased to publish some of these on the next few pages.

Occasionally in every field of endeavor there comes upon the scene an individual whose character, idealism, generosity, dedication and accomplishment stamp him as one of the great benefactors of his day.

Arthur Hastings Merritt is such a person. His devotion to all aspects of the progress of dentistry has been outstanding and his skillful and effective services to his profession and to his patients continues long after most practitioners would have sought the ease and comfort of retirement.

It is highly appropriate that the American Academy of Periodontology is honoring Dr. Merritt at its annual meeting in New York this fall. I know of no one more deserving of this honor, for he is a shining example of the truly professional man.

Bissell B. Palmer, D.D.S.
221 Fourth Avenue
New York 3, New York

I am delighted to hear that the American Academy of Periodontology is to honor Arthur Merritt at the annual meeting this fall.

Though over the years we have moved along different paths, my intimate association with him has meant much to me. His interpretation of his specialty is not that of one who at any time has "suffered amputation from the trunk." Throughout his professional career he has been a welding influence among divergent trends, never modifying his point of view to win favor. Men of of his strength of character, understanding and breadth of interest are rare indeed.

Today, when the limitations of specialization within specialties is being pointed to, paying homage to Arthur Merritt not only gives him his due; it reflects a timely attitude on the part of the Academy itself.

A. LeRoy Johnson

As a collector of early Americana Dr. Arthur H. Merritt speaks with patriotism, affection and wit on his favorite nondental subject: This Amazing America. The while he leads his audiences through a novel and delightful presentation of our country's history, this amazing American is himself a living legend.

Born and bred in the frugal New England tradition of plain living and high thinking, he transferred to New York in early manhood, here to invest his inherited riches. His inheritance? His riches? The only wealth that the rural New England of his youth could bequeath: character.

With this patrimony and little more—not robust health, nor a nest-egg of hard money, nor one influential friend—this amazing American became a New Yorker, dentist, periodontist, teacher, author, collector, historian, philanthropist. He was recognized and acclaimed by his professional colleagues all the way to the presidency of the American Dental Association. Everybody familiar with dentistry knows these things.

A very part of all he has become is what he started with: character, integrity in all his dealings, justice and tolerance in his decisions, generosity and faithfulness in his friendships. The writer came to know Dr. Merritt through his friend, the late Dr. Waldo H. Mork, who loved to tell how their long and treasured friendship came about. The circumstances were such as have characterized this man from the day he set foot in Manhattan as an applicant to enter dental school to this day when he is loved and honored throughout world dentistry.

There are two sides to every difference, and long ago in the affairs of the dental society, Dr. Merritt and Dr. Mork, without being personally acquainted, were found on opposite sides of a vexing question (what it was is lost in the passage of time). Dr. Merritt took a stand which he had reason to believe was right; Dr. Mork opposed it, for reasons he believed to be right. There was some bitterness in the conflict, and the occasion was forgotten. But when Dr. Merritt learned at last that Dr. Mork had been right, he sought him out, recalled the old disagreement, cleared up the honest misunderstanding, and struck a flame of friendship which has never dimmed.

Dr. Mork is gone, but his friend remembers him faithfully and generously with unpublicized gifts made in Dr. Mork's memory to Dental Health Service, our clinic which was Dr. Mork's absorbing interest for twenty-seven years. The friendship of these two

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men of character who met and measured each other long ago endures today through Dr. Merritt's philanthropy.

Let this brief account be one small candle in the brilliance which illuminates the life and work of this amazing American, Dr. Arthur H. Merritt.

Pauline E. Warner, Secretary
Dental Health Service
165 West 46th Street, New York 36, N. Y.

It is good news to hear that you are soon to honor and pay a tribute to my long-time friend, Dr. Arthur H. Merritt. Dr. Merritt is a neighbor of mine, and I have known him for over a quarter of a century. He is one of the most interesting and public spirited men I have ever known. It has always been a joy to meet him and talk with him.

Dr. Merritt has been a wide reader of worthwhile books. His autobiography is something to be proud of, as well as his continual contributions to publications throughout his life.

To honor such a man as Dr. Merritt inspires us all. Outside his regular work as one of the outstanding dentists in America, he is in addition a collector of paintings, etchings and Early American plates. These hobbies have kept him young. But I think his greatest distinction is being a great friend to all who have ever met and talked with him.

Sincerely yours,
George Matthew Adams
444 Madison Avenue
New York 22, N. Y.

Everyone at The Children's Aid Society is delighted to know of the honor to be paid by the American Academy of Periodontology to Dr. Arthur H. Merritt, one of our best and oldest friend.

It was largely owing to Dr. Merritt's interest and enthusiasm that the Society opened in 1907 at its 53rd Street Industrial School the first free school dental clinic in the country. Dr. Merritt brought to the project not only excellent training in dentistry, but a spirit for service which led many of his colleagues to volunteer their services to The Children's Aid Society for the dental care of children. The single clinic which Dr. Merritt was instrumental in founding has grown to ten clinics, which last year performed over 55,000 operations for 5,383 boys and girls, including special orthodontic and prosthodontic work.

Through the years, and down to this very day, Dr. Merritt has continued his interest and generous support of the Society's dental program; and the stress placed, under his guidance, on preventive dentistry in our first clinic is just as important today to the children we serve as it was in 1907.

The Children's Aid Society is proud to have this opportunity of joining with the American Academy of Periodontology in honoring Dr. Merritt, especially for his vital role in developing dental service to the city's children.

Arthur Huck
Executive Director, The Children's Aid Society
105 East 22nd St., New York, N. Y.

It is a pleasure to learn that your Academy at its annual meeting in New York this fall is honoring Doctor Arthur H. Merritt. I was associated with Doctor Merritt for thirty-five years and during that time the dental profession conferred upon him the highest honors it has to offer. He also received degrees from several of the leading universities. There is no need for me to comment about these honors, and his generous contributions to dental literature; or about the services he rendered to organized dentistry and to the formation of your Academy. These I feel sure will be described by others.

It is rather my purpose to tell you something about Doctor Merritt as a practitioner and as an individual. Fundamentally Doctor Merritt is a student. He is a great reader of anything pertaining to dentistry. He has collected a fine dental library. Self educated, he has accumulated a vast amount of knowledge, especially in the field of periodontology, as evidenced by the two books he has written. He is a gifted speaker and a fluent writer, as his numerous contributions to your magazine will attest. In practise, he is an excellent operator, and an expert as a periodontist. He is an able diagnostician. He has the faculty of inspiring confidence and has always enjoyed a very large clientele.

It is always a pleasure to be with Doctor Merritt. He is highly intelligent with a fine sense of humor. He is industrious, studious, religious, thrifty, generous, and kind. He is loyal to his associates and to his office staff. His hobbies, outside of dentistry, are the collecting of early American historical china and becoming familiar with the world's best poetry.

It has been my good fortune to be associated with Doctor Merritt for many years. During that time, I, with his other associates and with his office staff have acquired great admiration and affection for him.

Herbert S. Bailey, D.D.S.
116 DeSoto Road
West Palm Beach, Florida

It is a privilege to express my admiration of Dr. Arthur H. Merritt as a dentist and friend. He has had a long record of leadership of the highest order and at the same time has been one of the most lovable men of the dental profession.

His keen mind and good judgment combined with an unusual ability to express his thoughts clearly have made him a leader in the profession, one to whom we look with the highest esteem.

His interests have been devoted to the best type of dental teaching and dental service and yet he found time to serve the profession in many responsible positions of leadership including the presidency of various component societies to which he belongs and was president of the American Dental Association in 1940 during the Centenary Celebration of the founding of the first Dental School in 1840 and the beginning of organized dentistry. We were proud of Dr. Merritt as our presiding officer. He added much dignity to the occasion, as well as his efficient leadership.

Dr. Merritt has an unusual interest in art and literature. He has accumulated a great many pieces of beautiful historic paintings on blue china for which he had to build a fireproof vault in which to store them. He is a great reader of choice literature both poetry and prose and is able to quote from memory many beautiful verses and passages from various poets and writers.

To be a friend of Arthur Merritt is to get a glimpse of his genius and his character. His dauntless courage and patient persistence regardless of difficulties or handicaps have succeeded in bringing him to the pinnacle of achievement and success which he occupies in the history of dentistry and in the hearts of his many friends and admirers. Dr. Merritt has always looked for the good and the beauty in life and has been dedicated to a life of service and devotion to his profession, his family, and his country.

Edgar D. Coolidge, D.D.S.
636 Church Street
Evanston, Illinois

Repair of the Dento-Gingival Junction Following Surgical Intervention

by NICHOLAS R. MARFINO, D.D.S., M.S., BALINT J. ORBAN, M.D., D.D.S. AND FRANK M. WENTZ, D.D.S., M.S., PH.D.

IN 1950 Linghorne and O'Connell began a series of experiments. The first was "a study of soft tissue reattachment following surgical destruction of bone and periodontal membrane". Notches were made on the enamel surfaces of the canine tooth of a dog to mark the position of the gingiva and to be able to record clinically the depths of the pockets. They noted repeatedly "a reattachment of connective tissue of the gingiva to the tooth by means of a deposition of new cementum". They also observed "that, when reattached, the fibers ran in a parallel direction rather than the characteristic oblique direction to the tooth".

Linghorne and O'Connell (1951) in their follow-up experiment with dogs employed the same methods used previously but added autogenous bone chips to stimulate osteogenesis. Regeneration of the alveolar process in varying degrees was observed.

The same authors (1955) showed regeneration in surgically created "epithelized" pockets on dogs. After reflecting a soft tissue flap and removing some alveolar bone, a pack of zinc oxide and eugenol was placed in the pocket for three weeks to assure epithelialization of the pocket. A hypothesis was advanced to explain the observed regeneration as follows: "A proliferation of cells just beneath the epithelial attachment displaces the epithelium crownwise. The tooth is now exposed to new cells and is resorbed until differentiation of cementoblasts takes place. These lay down new cementum. After the resorptive process has progressed for some time the area of reattachment shows a continuous resorption of the tooth." This hypothesis was illustrated by the authors in a diagram (Fig. 1). Several investigators previous to Linghorne and O'Connell have experimentally investigated this problem.

Beckwith, Fleming and Williams (1927-1928) reported stages of regeneration of the artificially destroyed periodontal membrane, in guinea pigs and rabbits, animals with continually growing and erupting teeth. This was followed by a series of experiments on cats to see whether a like sequence of repair might be demonstrated in an experimental animal with teeth characterized by limited growth. The periodontal tissues were destroyed in an area remote from the gingival crevice. Regeneration of the periodontal membrane was again noted.

"A temporary reattachment of periodontal tissues in reimplanted dogs' teeth" was reported by Skillen and Lundquist (1934). Following this, the same authors (1937) did an experimental study of "periodontal membrane reattachment in healthy tissues of three dogs involving the gingival crevice". Pockets were produced by passing a sharp instrument under the labial gingival tissues separating them from the root. No bone was removed. Some pockets were treated with sodium sulfide and some were left untreated. Sections were removed after one week, two weeks and two months. The authors found, "at best, 2 mm. of connective tissue reattachment on the healthy tissues of dogs. Epithelium seemed to adhere to the root surface in deeper parts of the pocket".

Beube (1934) on dogs simulated the works of Beckwith, Fleming and Williams, but in addition he used devitalized heterogenous bone powder to act as a nidus for bone formation. Some production of new bone was observed. Further experiments

From Loyola University School of Dentistry, Chicago, and University of Buffalo, Departments of Periodontology and Oral Pathology.

This investigation was supported in part by Research Grant D-657, United States Public Health Service, from the Division of Research Grants, National Institutes of Health, Bethesda, Maryland.

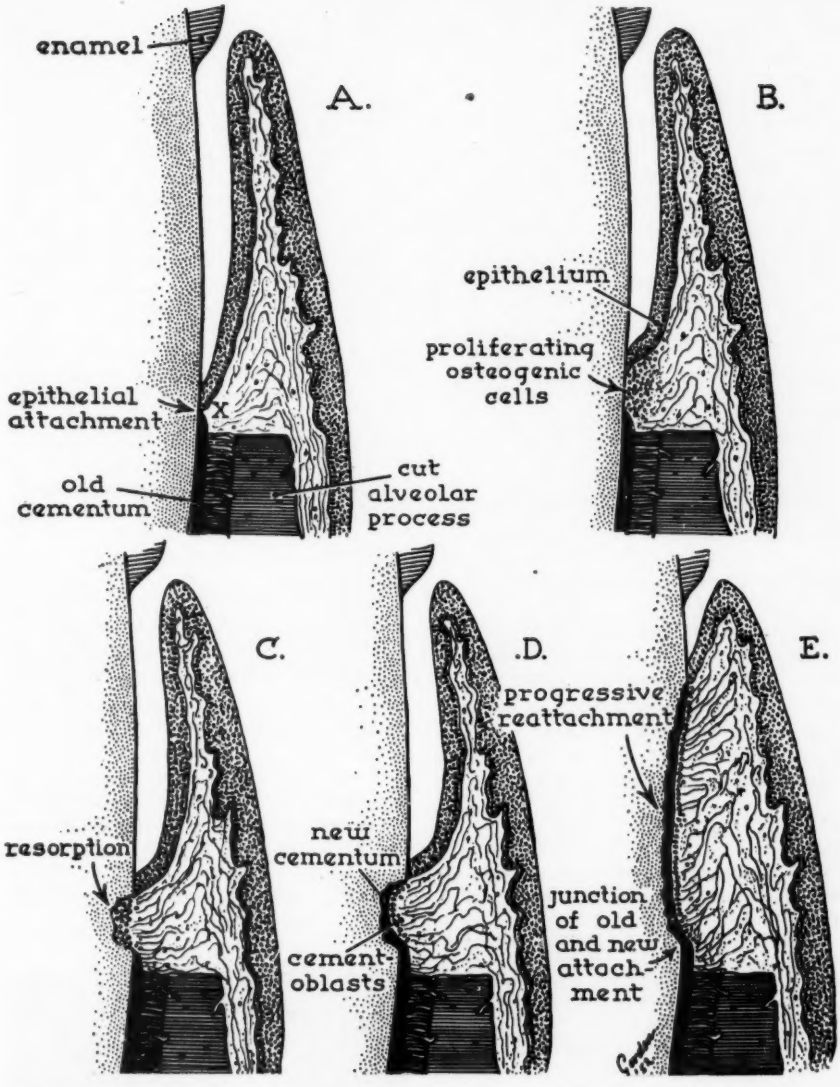

Fig. 1. Diagrammatic illustration explaining hypothesis advanced by Linghorne and O'Connell (1955) showing reduction of pocket by coronal movement of epithelial attachment ahead of connective tissue attachment.

The following is the original legend of this figure: "Schematic representation of the steps in reattachment according to our hypothesis (A.) Pocket. (B.) Cells at (X) have proliferated displacing the epithelium crownwise (C.) Tooth exposed to new cells is resorbed until differentiation of cementoblasts takes place (D.) Cementoblasts have differentiated and begun apposition of new cementum (E.) Appearance of reattached area of the reparative process has progressed for some time, showing continuous resorption of tooth."

(1941, 1942, 1947) with boiled cow bone showed histologic evidence of formation of cementum and periodontal membrane as well as bone.

Ramfjord (1951) took up the work started by Stone (1934) and Fish (1946) concerning surgically produced pockets on Rhesus monkeys. He demonstrated "both connective tissue and epithelial reattachment on root surfaces that had been exposed in an inflamed periodontal pocket and that removal of the epithelial lining of the pocket was necessary to obtain reattachment". He also noted that "the degree of inflammation in the area of healing appeared to determine whether the reattachment would be epithelial or connective tissue in type".

All these as well as other similar experiments have added to our knowledge of the biologic processes that take place in regeneration of the periodontal tissues.

However, especially the experiments of Linghorne and O'Connell of 1955 (see Fig. 1 Dia.) did not seem to correspond to the generally recognized regeneration process and therefore it seemed necessary to undertake experiments in the same arrangement as these authors have done.

MATERIALS AND METHODS

Ten young adult mongrel dogs, approximately two to three years of age, with full permanent dentition served as subject material. Surgical flap operations were performed on the maxillary canines in this investigation because of accessibility and lack of muscle tension in this area. The animals were anesthetized by nembutal administered either intravenously or subperitoneally.

Previous to the surgery at a point approximately 1 mm. incisally and parallel to the gingival margin a notch was cut, by means of a power-driven abrasive disc, into the labial enamel surface extending slightly into the dentin. This notch served as a point of reference from which measurements could be calculated after the tissues had been detached surgically from their original position.

The surgical flap was then made on the labial gingiva and mucosa directly above the notch, thus exposing the periosteal side of the alveolar bone. The incisions began immediately mesial and distal of the canine and extended upward and obliquely away from the long axis of the tooth for a distance of approximately 10 mm. in each direction. The flaps now had a base ranging between 25-30 mm., thus wider at its base than at its apex.

A section of alveolar bone approximately 7x6 mm. with the attached periodontal ligament was removed.

A second notch was then made on the tooth at the base of this exposed area. This notch served as a point of reference which limited the apical extent of this surgically produced "pocket," (Fig. 2). A pack of zinc oxide and eugenol was placed over the exposed area completely covering it, including the notch at the base. The pack was allowed to harden and the wound was sutured.

The pack was allowed to remain for three weeks and was then removed by teasing it out of the pocket. Clinical measurements of the distance to the gingival margin and the bottom of the pocket were made from the coronal notch.

The animals were sacrificed at the following intervals: 23 days, 36 days, 41 days, 51 days, 59 days, 76 days, 158 days, 160 days, 308 days, 331 days.

Histologic examination of the operated areas was performed.

CLINICAL FINDINGS

At the time of operation the artificial periodontal pocket, created by the detachment of the gingiva, periodontal ligament and removal of a 6x7 mm. section of alveolar bone averaged a depth of 10 mm. (which was measured in every case).

Two days after the removal of the pack, the most apical portion of the pocket could be visualized by gentle exploration and probing the area.

Weekly clinical examination, of the

Fig. 2. Clinical photograph of left maxillary canine with surgical flap retracted. Section of alveolar bone removed. Coronal and apical notches are seen.

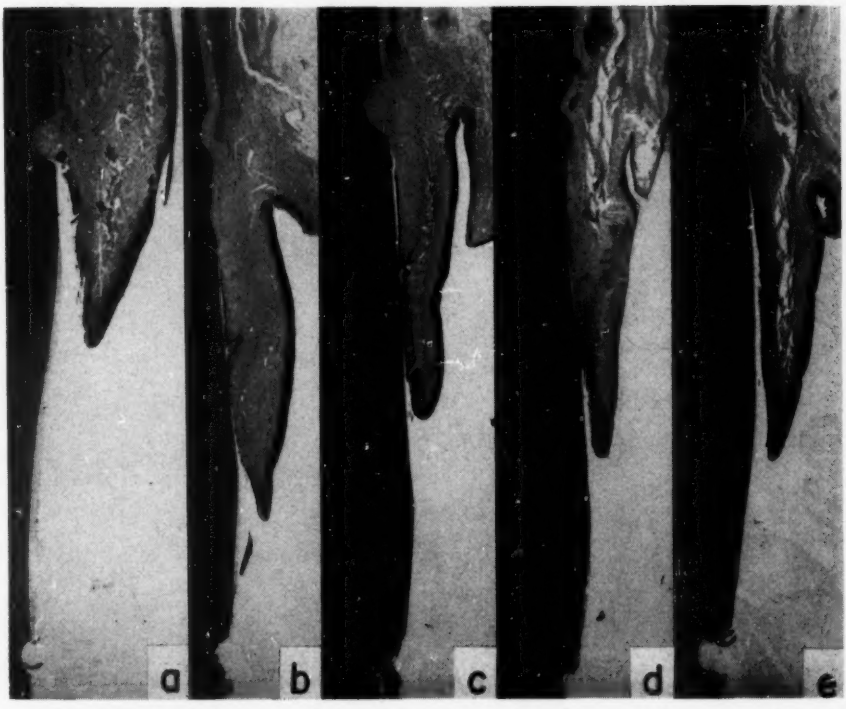

Fig. 3. Composite photomicrograph showing the relationship of soft tissues and alveolar crest to the tooth at various time intervals. (X10).
a) 36 day specimen, b) 51 day specimen, c) 76 day specimen, d) 158 day specimen, e) 331 day specimen.

animals, revealed a gradual recession of the soft tissue wall of the pocket in an apical direction. This recession progressed for about two months after the surgical detachment and then the position of soft tissues remained fairly constant.

At the fifty-first post-operative day the shrinkage of soft tissue averaged 3 mm. or about one-third of the length of the flap.

This recession of the soft tissue wall of the pocket led to a reduction of the depth of the pocket as measured from the coronal notch to the margin of the gingiva.

MICROSCOPIC FINDINGS

Twenty-three days. Histologic examination of specimens from an animal sacrificed twenty-three days after the surgical production of a periodontal pocket and two days after the removal of the pack revealed the bottom of this pocket to be located at its position at the apical edge of the notch in the root of the tooth. The pocket and this notch which marked the bottom of the artificial pocket were entirely free of remnants of the pack.

The wound area was epithelized, i.e., the epithelium completely lined the dental surface of the soft tissue pocket wall.

Thirty-six days. At thirty-six days the bottom of the artificial pocket was located at the coronal edge of the notch in the root of the tooth, Fig. 3a. A few remnants of pack and cellular debris were present in the pocket. The wound area was incompletely epithelized, i.e. the epithelium was disrupted and did not reach to the most apical part of the pocket. Here approxi-

mately ½ mm. of connective tissue remained exposed and the epithelium did not come into contact with the tooth.

Fifty-one days. At fifty-one days the bottom of the artificial pocket was located 4.25 mm. coronally to its original position at the time of surgery (Fig. 3b). The amount of reattachment is the sum of the amount of new connective tissue attachment plus new epithelial attachment.

There were no remnants of pack in the pocket. The wound area was epithelized, i.e., the epithelium lined the dental surface of the soft tissue pocket wall and was attached, at its most apical extent, to the tooth surface for a length of 1.5 mm.

The new connective tissue which was attached to the tooth surface in and coronal to the root notch was observed to be highly cellular in nature, most of the cells were fibroblasts although no definite collagenous fiber bundles could be distinguished. Some patent capillaries persisted although the endothelial cells were no longer swollen but instead appeared flattened. The apical crest of the alveolar bone is seen (Fig. 3b) to extend coronal of the notch, which means that this bone was formed after the surgical experiment. This area showed different appearance in different sections but active bone formation was in evidence in all. One such section is shown in Fig. 4—illustrating immature cellular bone extending coronally into and beyond the apical notch. The surface of this newly formed bone has an osteoid seam lined by osteoblasts. The surgically created notch in the dentin is lined by newly formed hard tissue, lined by cementoblasts.

Seventy-six days. At seventy-six days the bottom of the artificial pocket was located 3.75 mm. coronally to its original position at the time of surgery (Fig. 3c).

There were no remnants of pack in the pocket. The wound area was epithelized, i.e., the epithelium lined the dental surface of the soft tissue pocket wall and was attached, at its most apical position, to the tooth surface for a length of 2.00 mm., as the epithelial attachment. (In Fig. 3c a space appears between the epithelial attachment and the tooth. This is an artifact which was caused by the microscopic preparation.)

Fig. 4. Photomicrograph of 51 day specimen—left maxillary canine (X100).
Note: Immature cellular bone extending coronally into and beyond the apical notch. The surface of this newly formed bone has an osteoid seam lined by osteoblasts.

In the area of the root notch the connective tissue was organized into definite collagenous fiber bundles. For the first time a gingival group of periodontal ligament fibers could be distinguished.

One hundred and fifty-eight days. At one hundred and fifty-eight days the bottom of the artificial pocket was located 4.50 mm. coronally to its original position at the time of surgery (Fig. 3d).

There were no remnants of pack in the pocket. The wound area was epithelized, i.e., the epithelium lined the dental surface

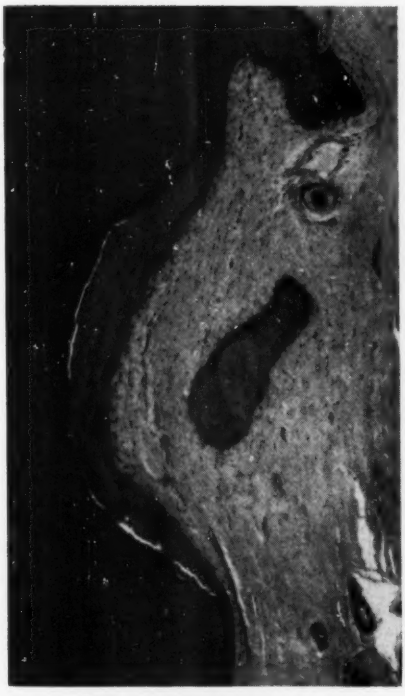

Fig. 5. Photomicrograph of 158 day specimen. Left maxillary canine (X100).
Note: The surface of the dentin at the side of the notch is covered by a fairly thick layer of newly formed hard tissue. The cementum is irregular at its coronal edge and some bone spicules extend into the notch of the tooth. The connective tissue filling the notch in the root of the tooth is relatively free of inflammation.

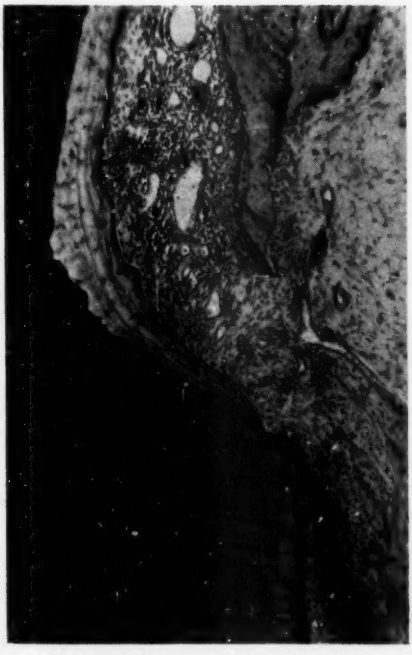

Fig. 6. Photomicrograph of 331 day specimen—right maxillary canine (X100).
Note: Apical end of the epithelial attachment and the newly formed bone-like hard structure covering the dentin in the notch. The tip of the alveolar crest extends into the notch. Some inflammatory cells are present.

of the soft tissue pocket wall and was attached at its most apical portion, to the tooth surface for a length of 2.00 mm. extending near the apical notch.

The connective tissue beneath the epithelial attachment and in the apical notch was differentiated into definite collagenous fiber bundles. The surface of the dentin at the side of the notch is covered by a fairly thick layer of newly formed hard tissue. The cementum is irregular at its coronal edge (Fig. 5) and some bone spicules extend into the notch of the tooth.

Filling the notch in the root of the tooth was connective tissue which was relatively free of inflammation.

Three hundred and thirty-one days. At three hundred and thirty-one days, the bottom of the artificial pocket was located 2.50 mm. coronally to its original position at the time of surgery (Fig. 3e). There were no remnants of pack in the pocket. The wound area was epithelized, i.e., the epithelium lined the dental surface of the soft tissue pocket wall and was attached, at its most apical extent to the tooth surface for a length of 1.75 mm. reaching to the coronal margin of the apical notch. In all other respects sections from this time interval closely resembled the sections from the one hundred and fifty-eight day specimen. A higher magnification of the coronal edge of the apical notch shows the apical end of the epithelial attachment (Fig. 6) and the newly formed bone-like hard structure covering the dentin in the notch. The

tip of the alveolar crest extends into the notch. Some inflammatory cells are present.

DISCUSSION

The repair of the dento-gingival junction. The timing, the degree, and the proportional amount of new connective tissue attachment and new epithelial attachment are significant findings of this experiment. The problem of reattachment of the soft tissues to the hard tissues of a tooth must be considered from two individual aspects before the mechanisms of regeneration may be clarified.

1. The attachment of the connective tissue elements to the tooth surface and alveolar margin.
2. The attachment of the epithelium to the tooth surface.

These two attachments then comprise the "dento-gingival junction". The connective tissue provides the mechanical resistance needed in such a junction of hard and soft tissues while the biological sealing of underlying structures against bacterial and chemical irritation is provided by the attachment of the epithelium to the surface of a tooth.

All findings indicate a relatively constant proportion of new connective tissue and new epithelial attachment. In viewing these problems a diagram might be helpful (Fig. 7).

The connective tissue had a range of attachment which measured from 1.25 mm. to 2.50 mm. from the apical portion of the notch in the root to the apical end of the epithelial attachment (Fig. 7-B1).

The epithelial atttachment was observed to be a cuff measuring in a majority of the specimens 1.50 mm. to 2.50 mm. in length (B_2) Fig. 7.

In this study the connective tissue was formed and attached to the tooth surface in the earliest specimens, but it organized slowly and could not be distinguished as definite collagenous fiber bundles until the

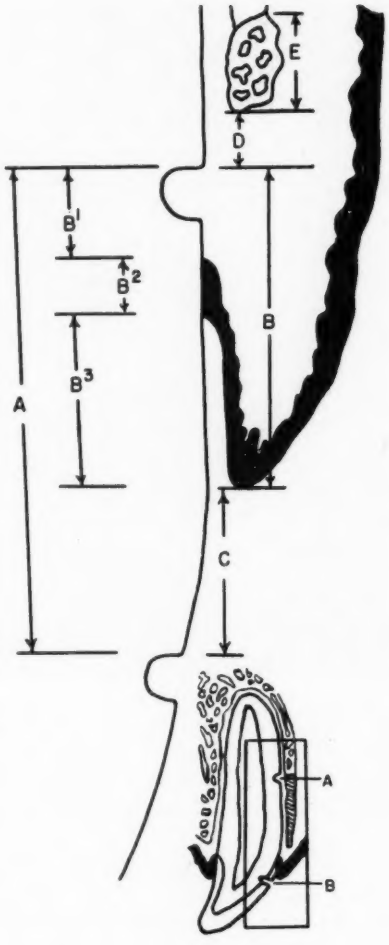

Fig. 7. Diagrammatic illustration—Key to microscopic measurements in present study.

A—Total distance between coronal and apical notches

B—Distance between apical notch and gingival margin

B^1—Amount of new connective tissue attachment

B^2—Amount of new epithelial attachment

B^3—Depth of pocket

C—Amount of atrophy

D—Distance between alveolar margin and apical notch

E—Amount of new bone added to alveolar crest

Insert: A—Notch in the root of the tooth (apical notch)

B—Notch in the crown of the tooth (coronal notch)

Fig. 8. Photomicrograph of 59 day specimen—maxillary left canine (X10).
Note. The position of the pack is in the notch—extending apically of the same.

seventy-sixth post-operative day. On the other hand, a definite epithelial attachment also may be observed as early as the twenty-third day after the surgical production of the artificial pocket.

It is interesting to note that definite orderly progressive stages of development could not be observed time-wise.

It appears that the variation in the pattern of healing exhibited is due to the presence of the pack in the pocket. It seems that if this pack was displaced from the notch in the root of the tooth early, this would allow a granulation tissue to form and organize into connective tissue before the epithelium had a chance to migrate down the pocket wall and prevent its organization. If the pack, however, was held in place for a longer period of time the epithelium then had more time to migrate down the pocket wall and attach itself to the tooth surface before the connective tissue could be formed. Figures 8 and 9 show such variation in the location of the pack. In Fig. 8 the pack is in the notch—extending even apically of same, however in Fig. 9, the pack has been displaced in coronal direction, leaving an area of the root surface coronal of the notch exposed to connective tissue as well as to the epithelium. The difference in the pattern of healing is obvious. Both of these pockets were 9 weeks old.

Comparison of findings with previous experiments. It has been pointed out in the review of the literature that such similar experiment was performed by Linghorne and O'Connell (1955). The findings in this study appear to be contradictory to theirs. However, certain procedures in this experiment differed from theirs. The variations in procedure, must be stated here, since the basis of explanation of the contradictory findings is dependent upon this comparison.

1. At the time when the artificial pocket was made, Linghorne and O'Connell used only one notch (in the enamel) as a point of reference. In their experiment to denote the most apical extent of the newly created pocket the cementum was scraped from the root surface of the tooth. The most

apical point of denudment of the cementum indicated the bottom of the pocket. This seemed highly inadequate since resorption which is constantly seen to take place in such a process of repair may easily destroy this point of reference.

Therefore in the present experiment in order to have an unmistakable point of reference marking the bottom of the surgically created pocket, a notch was placed in the root in addition to the crown as the second point of reference.

2. The figures recorded at the various time intervals by Linghorne and O'Connell were taken from the living animal by means of a probe calibrated in millimeters.

In the present experiment in addition to gross clinical measurements, microscopic measurements were made on histologic sections obtained from the animals sacrificed at the various time intervals. Realizing that a pocket does not always heal uniformly around a tooth, serial sections were made of the teeth and histologic measurements were taken. This was done by means of an eye-piece disc micrometer which was calibrated with a slide micrometer, measured in units of .01 mm.

3. No histologic sections before the nineteenth post-operative month were secured by Linghorne and O'Connell, nor were any microscopic measurements computed on the histologic specimens. However, only a hypothesis of what transpired during these post-operative nineteen months could be offered by the authors.

In the present experiment histologic sections were secured as early as 23 days post-operatively (2 days after removal of the pack) and at periods of 36, 41, 51, 59, 76, 158, 160, 331 days.

Fig. 9. Photomicrograph of 50 day specimen—maxillary right canine (X10).
Note: The pack has been displaced in a coronal direction leaving an area of the root surface coronal of the notch exposed to connective tissue as well as to the epithelium.

The hypothesis advanced by Linghorne and O'Connell suggests that during the healing process a progressive displacement of the epithelial attachment occurs in a coronal direction. Linghorne and O'Connell illustrated diagrammatically these progressive stages of healing according to their hypothesis (Fig. 1). This hypothesis therefore would indicate that the epithelial attachment must be closest to the apical

notch in the earliest specimens and furthest from this notch in the oldest specimens. These conclusions cannot be substantiated by the results of the present experiment. To the contrary, the epithelium was observed to be attached at its maximal coronal position from the apical notch in the younger specimens and progressively closer to the apical notch in the older specimens until at 331 days the epithelial attachment was found at the margin of the notch itself. This phenomenon might be interpreted that the epithelial attachment might have been once attached at a more coronal point and then in time shifted apically. However, the position of the epithelial attachment and some of the connective tissue reattachment was dictated by the position of the apical end of the pack Figs. 8-9.

SUMMARY AND CONCLUSIONS

This investigation represents a histologic and clinical evaluation of the healing mechanism which occurred following the surgical creation of periodontal pockets.

Ten young adult mongrel dogs with full permanent dentition served as subject material. The pockets, ten millimeters in depth, were produced by surgical flap operations and osteoectomy on the maxillary canines. Notches were made in the crown and root of the tooth to serve as reference points. The animals were sacrificed at the intervals: 23, 41, 51, 59, 76, 158, 160, 308, and 331 days.

The following were the major results obtained:

The formation of a new dento-gingival junction after the production of a 10 mm. artificially produced periodontal pocket is complete and consists of:

1. New attachment of connective tissue averaging 1.50 mm.
2. New attachment of epithelium averaging 1.75 mm.

These attachments occur at a point in the apical third of the distance between the two notches in the tooth. The depth of the remaining pocket however, diminishes but this is due to atrophy of the soft tissue flap. After the initial recession the depth of the pocket is more or less constant for the rest of the experiment. Microscopically no progressive coronal shift of the connective tissue and epithelial attachment ever occurred. These microscopic findings indicate, that reparative healing of the dento-gingival junction is complete and functionally acceptable in this experiment but with deformity, since a total regeneration of the original morphology did not occur.

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Inflammatory and Non-inflammatory Responses to Gingival Invasion by Microorganisms*†

by SOL HABERMAN, PH.D., *Dallas, Texas*

THE progress of gingival inflammation and the destruction of the surrounding tissues has been a subject of interest to many investigators. Reports on bacterial invasion as a cause of gingivitis have appeared since 1918. At that time, Turner and Drew[1] found cocci and diphtheroids in the gingivae from cases of "pyorrhea" and noted that rod forms were observed in the lymph channels of the infected areas. These findings were confirmed by Beckwith, et al[2,3] who photographically demonstrated bacteria in histological sections of inflamed gingival tissue. The validity of their histological evidence was supported by culturing bacteria from tissue aspirates in 161 cases of gingivitis. These cultures showed that streptococci and staphylococci were most commonly found, with only 34 of the 161 cases failing to demonstrate microorganisms. It was felt that the inflammation followed the directional influences of the structure of the connective tissue bundles and thus spread in a line parallel to the gingival crevice. The invaders penetrated the lymphatics and by following the line of drainage, extended to the periodontal membrane toward the apex and then to the root. James and Counsell[4] and Weinmann[5] have examined sections of teeth and supporting structures and observed that inflammatory infiltration follows the course of blood vessels and lymphatics through the gingival connective tissue to the bone. Box[6] confirmed these tissue findings and in addition, described "cockle-bur" like masses and threads of fungus filaments in the "cementum cuticle," subgingival calculus, and the partially calcified regions which extend into the pocket. The studies by Box[7] on the tissues of the gingivae involved in pockets showed the "brush-like and cockle-bur" formations in close proximity to a break in the epithelial continuity of the pocket lining, but these filaments were not observed in the intact epithelium.

During our recent study on bacteremia produced by gingivectomy,[8] the excised gingivae were preserved in formalin for histological study. These were examined microscopically to see if an explanation could be found for the high incidence of bacteremia following dental surgery. When the gingivae were sectioned and stained by

*Department of Bacteriology, Baylor University, College of Dentistry. Presented as an Exhibit, "Bacteremias Following Dental Surgery" at the 1958 Meeting of the American Academy of Periodontology, Baker Hotel, Nov. 6-8, Dallas, Texas.

†This investigation was supported in part by a research grant D-606, National Institute of Dental Research, Public Health Service, Bethesda, Maryland.

the Brown and Brenn modification of the Gram stain, nidi of microorganisms in the epithelial and connective tissue areas were observed. These observations led to this more thorough examination of excised gingivae from cases of gingivitis.

METHODS

The gingival strips obtained from patients undergoing gingivectomy were placed in containers of 10% formol saline for fixation. These patients were part of the study on the incidence of bacteremias resulting from gingival resection. Pieces of the excised gingivae were selected which showed normal as well as inflamed areas. These were imbedded in "tissue mat" and prepared for serial sectioning with a rotary microtome. The cuts were made in the transverse plane to the strip. The thickness of each section was approximately 5 microns. The tissue ribbons thus obtained were divided so that a set of three consecutive sections was mounted on separate slides after each 15-20 cuts. The first of each of the sets of 3 was stained by the haematoxylin-eosin method;[9] the second section was stained by the Brown and Brenn[10] modification of the Gram stain and the third was stained by the Grocott modification[11] of Gomori's methenamine-silver nitrate technic which is excellent for the staining of many fungi.

In the Brown and Brenn method, the tissue was stained with a 1% crystal violet that was alkalinized with 5% sodium bicarbonate. A relatively short treatment with Gram's iodine was used to mordant the dye before the decolorizing process was begun. The first decolorizing was accomplished with an ether-acetone solution which rapidly dissolved out the excess crystal violet. A counterstain of basic fuchsin was applied and the tissue was decolorized a second time in a picric acid-acetone solution until the sections were yellowish pink. After mounting with "permount" the microscopic examination showed the Gram positive bacteria as blue, Gram negative bacteria as red, tissue nuclei as red, and the other tissue elements in shades of yellow.

The Grocott modification of the Gomori stain starts with the oxidation of the tissue section with 5% chromic acid followed by the removal of the residual chromic acid with a wash of 1% sodium bisulfite. The sections thus prepared were submerged in working methenamine-silver nitrate solution for 30-60 minutes at 58° C. When the sections turned brown, the slides were rinsed in distilled water and examined. The fungi appeared dark brown at this point before the tissue was toned with 0.1% gold chloride. The unreduced silver nitrate was removed with 2% sodium thiosulfate and a counterstain of light green was applied. A well stained slide showed the fungi to be black with the inner parts of mycelia and hyphae in shades of rose. The background tissue was pale green and mucin was colored taupe to dark gray. While Grocott states this method is not specific for the cellulose-chitin aggregates characteristic of fungi, it may be successfully used to stain Histoplasma, Coccidioides, Cryptococcus, Candida and others. It is of interest to note that this method fails to stain clearly the Actinomyces. The clubbed ends show little darkening and the threads do not stain like other fungi. The Brown and Brenn method stains both Nocardia and Actinomyces more clearly and this stain is preferred for these organisms.

RESULTS

Examples of the tissue findings are presented in Figures 1 and 2. In some areas of the sectioned gingivae, the cellular arrangements appeared normal when stained by the haematoxylin-eosin method. However, careful examination under the oil immersion lens would show occasional areas of increased density near the blood vessels in the connective tissue. When the consecutive tissue sections were stained by the modified Gram stain or the Gomori's methenamine-silver nitrate method, bacteria and fungus-like filaments were observed. Figure 1a shows a section of gingival connective tissue with apparently normal configuration. The flattened blood vessel lying vertically in the center of the field contains erythrocytes in the lumen. The surrounding tissue demonstrates no microscopic evidence of inflammatory response in the area. When the Gram stained consecutive section is examined, Figure 1b, the ends of the flattened capillary shows masses of Gram positive organisms which appear to be in the capillary wall. The distortion resulting from this stain make such observations difficult to demonstrate clearly. However, the Gomori stain of Figure 1c of the third of the consecutive sections, clearly shows the invasion of the capillary wall by microorganisms. The types of microorganisms present are not clearly defined but filamentous rods are present.

In Figure 2a, an haematoxylin-eosin stain of gingival connective tissue is shown. This area was located near the gingival attachment and shows an area of chronic to subacute inflammation with mononuclear cells of the lymphocyte and plasma cell types present. The sectioned blood vessel

located at the upper right of the area serves as a marker for this set of three consecutive sections. The inflammatory cells may be seen accumulated around the vessels running diagonally in the photograph. The Gram stain of the second section of the series, Figure 2b, shows the accumulations of many Gram positive cocci, bacilli and filaments located perivascularly. Again, the distortion in the Gram stain makes definitive statements on observations difficult. In Figure 2c, the Gomori stain of the third section with the marker vessel in the upper right of the picture, the perivascular infiltration and invasion of the vessel walls can be seen. The invasion by the microorganisms seems to be following the course of the vessels through the gingival connective tissue. The microorganisms seem to be of the coccus and rod forms with many filaments present.

The observations exemplified by Figures 1 and 2 are good examples of many observations made from the gingival strips obtained from patients with periodontitis that required gingivectomy.

DISCUSSION

It would seem obvious from the consecutive sections of Figures 1 and 2 that the demonstration of microorganisms in tissues should be done by methods other than haematoxylin-eosin. Although this stain is a technic of choice when one desires to study cellular structure in tissues, its failure to reveal the fungi and bacteria of this study is evident. The presence of these microorganisms in large numbers suggests that they have been in the tissue for some time. The accumulations of these around the vessels (Fig. 1c) with no inflammatory response is unusual, especially when one expects and observes an accumulation of phagocytic and other inflammatory cells at the site of invasions in other parts of the body.

The growth of microorganisms in gingival connective tissue, especially as perivascular invaders, might account for emboli in the lymphatic system and these in turn could be seeded into the blood stream. The surgical trauma of gingival resection may cause such a seeding directly into the blood stream or into the lypmphatics to give the bacteremias that Vargas, Collings, Polter and Haberman[8] found during this procedure.

The area represented by Figure 2 shows the same type of microorganisms observed in Figure 1, but in this case they seem to have elicited a chronic inflammatory response. Such findings were usually seen in areas near or at the epithelial attachment and extending into the periodontal membrane and connective tissues. Both the microorganisms and the cellular infiltration extended along the involved vessels to the deeper tissues. In the areas of invasion involving the connective tissues and their vessels, the non-inflammatory response was observed when the tissues were studied by proper stains. When the sections showing such a non-inflammatory invasion were carefully studied with the high power and oil immersion objectives of the microscope, small areas of density around the vessels and in the involved connective tissue could be observed with the haematoxylin-eosin stains. These gingival invasions may well explain the lack of observations of inflammation by the usual stains, yet the tissue continues to show clinical gingivitis. The progress of such infections could cause further fibrosis of the vessels, thus embarrassing the blood supply and leading to deeper damage and involvement which may affect the periodontal structures and cause alveolar crest resorption.

It is apparent that the findings of Beckwith,[2,3] Vargas, et al,[8] and Haberman, et al,[12] clearly demonstrate the fact that microorganisms do invade the gingival tissues, using the lymphatics and the perivascular route as the means of penetration. The perivascular infiltration observed by James and Counsell,[4] Weinmann[5] and Box[6] were the results of the tissue response to such infections. The failure to use the more specialized stains such as the Gram and

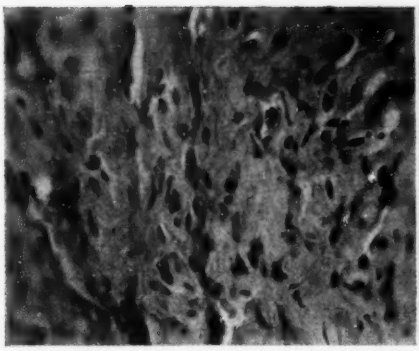

Fig. 1a. Serial section showing connective tissue of gingiva. Note the capillary lying vertically in the center. Area shows no microscopic evidence of inflammation. Hematoxylin-eosin stain; x675.

Fig. 2a. Serial section of gingiva showing increased perivascular fibrous tissue and the presence of chronic inflammation. Hematoxylin-eosin stain; x675.

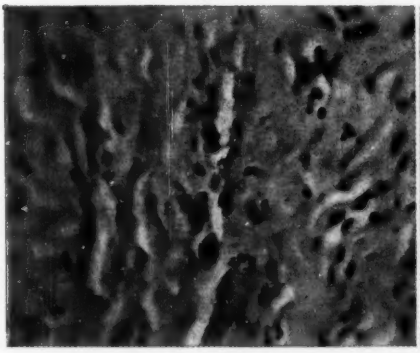

Fig. 1b. Consecutive section showing aggregates of microorganisms invading the wall at each end of the vertical blood vessel. Brown and Brenn modification of Gram stain; x675.

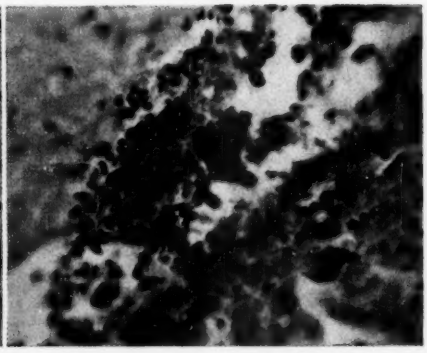

Fig. 2b. Consecutive section showing bacteria along the course of the capillaries. Note the filaments and cocci. Brown and Brenn modification of Gram stain; x675.

Fig. 1c. Next section of same capillary showing the perivascular invasion by microorganisms. Gomori's methenamine-silver nitrate method; x675.

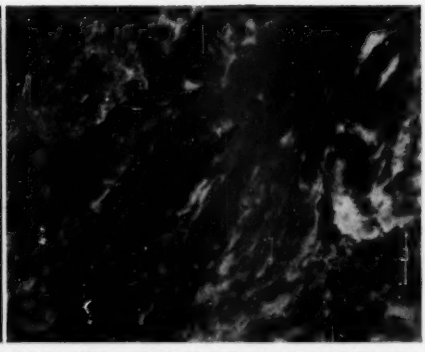

Fig. 2c. Next section of series showing perivascular infiltration and invasion by filamentous and other bacteria. Gomori's methenamine-silver nitrate method; x675.

silver-nitrate methods probably account for the failure of many investigators to find the invading microorganisms in periodontal diseases. For example, Fish[13,14] and Bibby[15] as well as others have expressed the view that bacteria produce their damage in periodontitis without invading the tissues themselves. The production of such damage is accomplished in some vague manner by enzymes and toxins elaborated by the bacteria in the periodontal pockets. The sequence of photographs presented in Figures 1 and 2 makes it difficult for this author to accept such a view. Figures 1c and 2c clearly show that microorganisms do invade the soft tissue side of the periodontal pocket. It appears that the areas invaded failed to show chronic inflammation in some instances and the organisms apparently migrate to the deeper tissues by perivascular infiltration.

The author expresses his appreciation to Mr. E. M. Grube of the Department of Visual Education for his valuable assistance.

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THE AMERICAN ACADEMY OF ORAL PATHOLOGY

At the 13th Annual Meeting of the American Academy of Oral Pathology held 17-19 April 1959 at the Mayo Auditorium, University of Minnesota, the following slate of officers was elected: President, Col. Joseph Bernier; President-Elect, Capt. Robert Colby; Vice President, Cdr. Henry Scofield; Secretary-Treasurer, Dr. Robert J. Gorlin; Editor, Dr. Donald Kerr; Council, Dr. Victor Halperin, Dr. Nelson Large, Dr. Irving Meyer, Dr. Hamilton B. G. Robinson, Dr. William Shafer, Dr. Richard Tiecke.

American Board of Oral Pathology Examiner—Dr. Charles Waldron.

Effects of Certain Factors on Bacteremias Resulting from Gingival Resection*

by BENJAMIN VARGAS, M.SC.D.,† D.D.S.; C. KENNETH COLLINGS, D.D.S.; LUCY POLTER, M.T., AND SOL HABERMAN, PH.D., *Dallas, Texas*

SINCE the reports of O'Kell and Elliot[17] in 1935 and Elliot[10] in 1939 showed that bacteremia frequently followed dental surgery, a number of investigators have observed bacterial invasion of the blood stream following various dental procedures. These included observations following routine prophylactic treatments, gingival massage, rocking of teeth before extraction, and the effects of multiple and single extractions. The presence of microorganisms in the blood stream has been attributed to the dissemination of bacteria from areas of infection at the apices of teeth, periodontal tissues, and from gingival crevices. These reports tend to correlate the incidence of bacteremia with a variety of factors which include the degree of periodontal disease, type of anesthetic used, and the amount of trauma produced during surgery. For example, Richards[19] recovered *Streptococcus viridans* from the blood stream of 3 out of 17 patients whose gingival tissues had been massaged for 10 minutes and Murray and Moosnick[16] reported that the chewing of paraffin for 30 minutes resulted in a 55% incidence of bacteremia from 336 patients who had various degrees of caries and periodontal disease. Robinson, et al.,[20] studied the incidence of bacteremia in 559 dental operations which included extractions, tooth rocking, cyst enucleation, alveolectomy, removal of impacted teeth, mastication, radicular treatment, and a periodontal treatment consisting mainly of 10 minute scaling on the four mandibular incisors. In this latter procedure they reported 5 positive blood cultures out of 72 cases. Similar observations by Cobe[7] showed an incidence of bacteremia after such procedures as prophylaxis, chewing a hard substance, chewing a soft substance, brushing and exodontia. He found a 40% incidence of bacteremia occurred during the "standard accepted method of prophylactic treatment." Blood cultures on patients having extractions showed 35.2% positive cultures, brushing 24.2%, and the chewing of hard candy yielded bacteremia in 17.4%. None of the 200 patients who had been chewing gum only demonstrated positive blood cultures. In trying to evaluate the role of periodontal disease in the incidence of bacteremias he concluded that factors other than the number of bacteria, such as enzyme activity or lack of intact epithelium, may account for bacterial penetration. It is of interest to note that Coffin and Thompson[8] found the highest incidence of bacteremias occurred after exodontic procedures among patients suffering from periodontal disease. They offered the suggestion that infection of the supporting soft tissues may be more important than infection of the bone.

The work of Fish and Maclean[12] and Fish[11] indicated that microorganisms do not invade the tissues beyond a necrotic nidus where they are protected from leucocytes. It was the opinion of Fish[11] that gingivectomy was not attended by a bacteremia unless the knife was blunted and pulled on the tissue. He found organisms only in ulcers but did not report them in the deeper tissues. These findings were contrary to the 1925 and 1927 reports of Beckwith, et al.,[1,2] wherein they described the presence of bacteria in the periodontal

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*This investigation was supported in part by a research grant D-606, National Institute of Dental Research, Public Health Service, Bethesda, Maryland.

†Presented in part as the thesis for the Master of Science in Dentistry, Baylor University, College of Dentistry.

tissues. Additional findings of this type were made by Box[3, 4] who described a fungus-like organism which was designated as *Leptothrix falciformis*.

Although the current literature in the field of periodontology refers to the possible occurrence of bacteremia following gingival resection, little has been found concerning the incidence of such bacteremia and the factors that may influence it. Periodontal disease, however, has been considered as a contributing factor to the incidence of bacteremia following exodontal procedures.

MATERIALS AND METHODS

Method of selection for patients. This investigation was made on patients in the Department of Periodontology of Baylor Dental College. The diagnosis of periodontitis and the need for gingival resection was determined after clinical and roentgenographic examination. All patients in this study received a dental prophylaxis 5 to 7 days before the tissue resection was performed. The dental prophylaxis previous to the main operation was intended to improve the clinical conditions of the oral tissues, reduce the possibilities of post-operative infection and enhance the healing process. When a full-mouth gingivectomy was needed, it was performed in two stages, two to three weeks apart. This afforded an opportunity of obtaining two blood samples from the same individual under similar conditions.

Methods of sulcus and mobility measurement. Before the gingivectomy was to be performed, the patient's teeth were examined individually and data was recorded as to mobility and depth of gingival sulcus. Mobility was determined by rocking each tooth between the two forefingers, buccally and lingually or labially and lingually, according to the position of the tooth on its arch. A firmly attached tooth was given a value of 0; a slightly noticeable mobility of the tooth was recorded as 1. Extreme mobility, probably requiring extraction, was valued at 3; and a mobility which was not extreme, yet easily inducted by the fingers, was valued at 2. Each tooth was examined for sulcus depth on its four aspects, namely, mesial, distal, lingual and buccal or labial, using a periodontal probe, and the depth was recorded in millimeters.

Cultures of gingival sulcus and surface. Using the same periodontal probe employed in measuring each of the pockets, blood agar plates were streaked for culture from the material obtained on the probe. Cultures were also made of the surface gingival tissues involved by running sterile cotton swabs on both the lingual and buccal aspects of the gingivae. These were observed for surface bacterial flora. The sulcus and gingival cultures were observed for a minimum of 48 hours, and the types of organisms present were identified.

Collection of blood cultures. The time of collection of the 114 blood samples is shown in Table 1. These ranged from 20-130 minutes after the initial surgical incision. This time usually coincided with the completion of the operation, or fell within ten minutes before the surgery was finished. A blood sample of 18-20 cc. was obtained from each patient and was divided into three portions as follows: 3 cc. were added to a flask containing 7 cc. of sterile 3 percent sodium citrate solution. This was then mixed with 50 cc. of melted (45-50° C.) blood agar base and three pour plates were prepared for a quantitative count of the bacterial colonies that might grow during incubation. The remaining portion was divided equally between two bottles; one containing 120 cc. of 3 per cent trypticase soy broth for aerobic culture and another containing 150 cc. of semi-solid trypticase soy agar for anaerobic culture. The cultures were incubated at 37° C. and observed for growth every 24 hours for ten days. When no growth was observed at the end of this incubation period, the culture was discarded as negative. All positive growths were subcultured and the organisms identified. In addition, a sensitivity test was performed on the isolated organisms by the infiltrated paper disc technic.[38]

RESULTS

One hundred and fourteen blood samples were collected from 89 patients. In 25 cases, a second blood sample was taken when the patient returned for a second surgical procedure. As may be seen in Table 1, 28 or 24.5% of the total 114 samples showed bacterial growth. Of the 25 patients from whom two samples were obtained, 17 yielded negative cultures in both instances, 6 gave positive cultures on both occasions and 2 showed growth from the first sample only.

The relationship of time of collection of the blood to the incidence of positive cultures is also shown in Table 1. It can be seen that a 25-30% incidence occurred until after 60 minutes had elapsed between the initiation of the surgical procedure and the collection of the blood sample. The highest incidence was at the 46-60 minute period when a 31% incidence occurred. After 60 minutes the occurrence of positive blood cultures dropped. This decrease correlates well with the fact that the surgical

TABLE I

Relationship of time of collection of blood sample during gingivectomy to incidence of findings on culture.

Time Interval from Initial Incision to Collection of Blood Sample	*Total Number of Blood Cultures*	*Number of Negative Blood Cultures*	*Number of Positive Blood Cultures*
20-45 Min.	47	35	12 (25.53%)
46-60 Min.	32	22	10 (31.25%)
61-130 Min.	35	29	6 (17.14%)
Totals	114	86	28 (24.56%)

P=0.4143

The formula for Chi square calculations in a 2 x 2 table is:

$$X^2=\frac{N(ad-bc)^2}{(a+b)\,(c+d)\,(a+c)\,(b+c)}$$

where:

a, b, c, and d are observed frequencies in the 2 x 2 table and N is the total number of cases observed.

	+	−	
	a	b	a+b
	c	d	c+d
	a+c	b+d	N

df=1

Therefore: for categories 20-45 minutes and 46-60 minutes of Table I.

	Positive	*Negative*	*Total*	*% Positive*
20-45 Min.	12	35	47	25.53%
46-60 Min.	10	22	32	31.25%
Totals	22	57	79	5.72% Difference

$$X^2=\frac{79\,(350-264)^2}{(47)\,(32)\,(57)\,(22)}=0.3098$$

The demonstration of $X^2=0.3098$ with P (probability) less than 0.50 indicates that the difference between the above mentioned categories is not significant.

For categories 20-60 minutes and 61-130 minutes.

	Positive	*Negative*	*Total*	*% Positive*
20-60 Min.	22	57	79	27.86%
61-130 Min.	6	29	35	17.14%
Totals	28	86	114	10.72% Difference

$$X^2=\frac{114\,(342-638)^2}{(79)\,(35)\,(86)\,(28)}=1.5002$$

The demonstration of $X^2=1.5002$ with P greater than 0.20 indicates that the difference is not significant.

TABLE II

The incidence of positive blood cultures during dental "prophylaxis"

Time of Collection after Start of "Prophylaxis" Procedure	*Negative*	*Positive*	*Total*
15 Minutes	26	5(16.1%)	31
After 20 Minutes	24	0	24
	50	5	55

$$\text{Chi}^2 \text{ Test of Significance:} = \frac{55(26\times0-24\times5)^2}{31\times24\times50\times5}$$

$X^2=1.0645$ $P=>0.30$ Indicates the difference is not significant.

procedure was usually being completed at that time, and a minimum of additional trauma was produced.

When the data was submitted to the Chi square test it showed that time of blood culture collection was not significant. However, there was a trend toward significance.

A comparison of the depth of the gingival sulcus and the mobility of teeth in both positive and negative cases was made in Table 3. There was no significant difference between the depth of the sulcus and the tooth mobility of those cases yielding a positive culture as compared to those yielding a negative one. Even when the maximum depth and mobility were considered, no significant variation was found.

The incidence of positive blood cultures is compared to the extent of the operation in table 4. The percent of positive cultures was slightly higher in the group having gingivectomies on more than one quadrant at a time. However, in 8 cases in which an osteoectomy or osteoplasty was performed in addition to the gingivectomy, no positive cultures were obtained.

Table 5 correlates the incidence of positive blood cultures to the quadrant involved in the operation. The figures show the largest percent of positive cultures when the lower quadrants were involved and the smallest when only the upper was treated surgically. It is of interest to note that when the blood sample was taken after gingivectomy on both upper and lower quadrants, the percent of positive cultures approaches that for the cases involving just the lower.

The frequency of occurrence of the various organisms isolated from the gingival sulcus, the gingival surface, and the blood cultures is shown in Table 6. *Streptococcus viridans* was the most frequently isolated microorganism, appearing in all

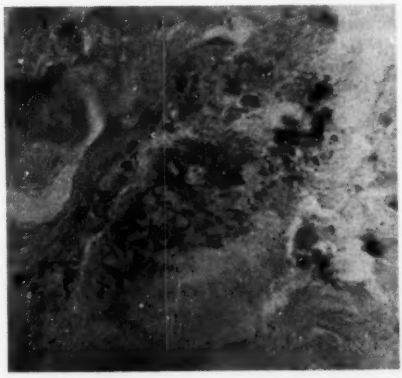

Fig. 1. Tissue section prepared from gingival resection by Brown and Brenn modification of the Gram stain. Note the area of increased density in the connective tissue which shows the Gram positive filaments, rods, and coccus-like organisms. Approximately 970X.

TABLE III

Relationship of depth of gingival sulcus* and mobility of teeth to blood culture results

	Number	*Percentage*	*Average Depth of Gingival Sulcus in mm.*	*Average Maximum Depth of Gingival Sulcus in mm.*	*Average Tooth Mobility*	*Average Maximum Tooth Mobility*
Positive Blood Cultures	28	24.56	3.07	6.14	.95	1.82
Negative Blood Cultures	86	75.44	2.72	6.05	.94	1.84
Totals	114	100	...	...	..	...

*As measured from the margin of the free gingiva to bottom of sulcus.

cultures of the gingival sulcus, in 99.11 percent of the gingival surface cultures and 53.57 percent of the blood cultures. Such a direct relationship was not shown for all the organisms listed. In the group of 6 patients yielding a positive blood sample on two different occasions, the organisms isolated were the same in only 2 instances.

Table 7 shows the in vitro sensitivity of the various organisms isolated from blood culture to 9 antibiotics. All of the organisms found were highly sensitive to penicillin, chloromycetin, erythromycin and novobiocin in this study.

Microscopic sections prepared from the excised gingival tissues, and stained by the Brown and Brenn[5] method of the Gram stain, showed bacteria which seemed to be located inside the tissues themselves. As shown in Figure 1, rods, filaments and cocci were present.

DISCUSSION

The results obtained in this investigation indicate that the incidence of bacteremia following gingivectomies on patients previously given a dental prophylactic treatment is 24.56% or more. Currently, reports on the incidence of bacteremia after oral surgery procedures emphasize the theory that "rocking" of the tooth before extraction produces a piston-like action which pumps the bacteria into the blood stream. However, in contrast to exodontia, gingivectomy is a procedure where "rocking" is minimal and in some instances nil, yet the incidence of bacteremia approximates that reported during exodontic procedures. Recently, Coffin and Thompson[8] reported 21.25% incidence of bacteremia after exodontic treatment of 950 patients afflicted with periodontal disease. They suggested that the infection of the soft tissues surrounding the tooth may be of more importance in the incidence of bacteremia than infection of the bone. The results of our investigation seemed to confirm this suggestion.

When the cultures were compared as to time of collection, it was found that those samples collected 20-60 minutes after the initial surgical incision showed a higher frequency of positive findings than those blood cultures collected after 60 minutes. When the Chi square test for significance is applied to these comparative data, the differences between the sets were not significant. However, in the group collected 20-45 minutes after the initial incision a value of $X^2 = 0.1252$ was obtained, while in the 46-60 minute group $X^2 = 0.1847$, and finally the 61-130 minute group gave a X^2 of 1.0341. This increasing value of X^2 from the shorter to the longer time interval shows a tendency toward significance. The important relationship of the time of collection of the blood culture to the start of exodontic procedures has been recognized;

TABLE IV

Relationship of extent of periodontal surgery to blood culture results

Extent of Operation	*Total No. of Blood Cultures*	*Negative Blood Cultures*	*Positive Blood Cultures*
5-8 Teeth (1 Quadrant)	44	35	9 (20.45%)*
9-16 Teeth (2 Quadrants)	69	51	18 (26.08%)*
17-32 Teeth (4 Quadrants)	1	0	1
Totals	114	86	28 (24.56%)

*P=>0.50 for 5-8 teeth vs. 9 16 teeth.

Chi square calculations for categories 5-8 teeth and 9-16 teeth.

	Positive	*Negative*	*Total*	% *Positive*
5-8 Teeth	9	35	44	20.45
9-16 Teeth	18	51	69	26.08
Totals	27	86	113	5.63% Difference

$$X^2=\frac{113(459-630)^2}{(44)\ (69)\ (27)\ (86)}=0.4468$$

The demonstration of $X^2=0.4468$ with P=>0.50 indicates that the difference is not significant.

however, in our studies on gingivectomy this factor was not a major influence. In exodontia, the bacteremia is reported to be of short duration immediately following the extraction. The data presented in Table 1 indicates that the bacteremia incident to the prolonged surgery of gingivectomy may be of longer duration, often demonstrable up to 2 hours after the initial incision (17.14%). It seems that as long as surgical trauma is being inflicted, bacteremia continues at a somewhat constant rate.

In the control study, 55 patients were used to determine when bacteremias are most common during dental "prophylaxis." The patients were divided into two groups, those from whom the blood was collected 15 minutes after the start of the procedure, and those whose blood was collected 20 or more minutes after the treatment had started. It was found that 5 out of 31 patients or 16.1% gave positive blood cultures when the sample was taken 15 minutes after beginning the "prophylactic" while no positive cultures were obtained from 24 patients whose blood sample was taken after the 20 minute period (Table 2). These findings differed somewhat from the data obtained from gingivectomy. In the prophylactic treatment the bacteremias were fewer, occurred earlier, and apparently lasted for a shorter time. This finding again correlates with the time during which tissue trauma and gingival manipulation is greatest. Thus, one might infer that the less the trauma and disturbance of the gingivae, the fewer bacteria will be found in the blood stream. Consequently, methods of judging the condition of gingivae might be an index as to the likelihood of bacteremias resulting from manipulation and surgery.

At the outset of this investigation, it was felt that the depth of the gingival sulcus would be a factor in the incidence of bacteremia following the removal of the gingival tissues in patients with periodontal disease. This assumption was based upon the findings of Burket and Burn,[6] which showed that the gingival sulcus was a source of invasion of the blood stream by bacteria during exodontia. It was assumed, therefore, that the greater the depth of the gingival sulcus the greater the concentration of bacteria and thus the larger the inoculum available for bacteremia. However, in our study on gingivectomy it was found that after measuring the depth of the sulcus on the four aspects of each of the teeth involved and taking an over-all average, as well as a maximum depth average, the results obtained do not seem to support this assumption (Table 3) for gingival surgery. It is of importance to emphasize that each of these patients was treated previously with a dental prophylaxis before the tissue resection was performed.

Tooth mobility was another factor that was considered at the outset of this investigation as an influence on the occurrence of bacteremia. One of the explanations for bacteremia following exodontia has been the rocking technic often used during extraction.[10,18] This "pumping action" could force bacteria into the ruptured blood vessels and into the circulation. Consequently, a greater mobility might be expected to produce a greater incidence of bacteremia. The results shown in Table 3 regarding average mobility and average maximum mobility compared to positive and negative blood cultures indicate that mobility had no influence on the incidence of positive blood cultures. The data shows almost identical mobility calculations for both the positive and negative blood culture groups. These findings again emphasize the differences in factors influencing bacteremia following exodontia as compared to gingivectomy.

The extent of surgery has been reported by many as an influence on the incidence of bacteremia in exodontia. The explanation offered is that the greater the number of damaged blood vessels the greater the opportunity through which the bacteria can invade the blood stream. Table 4 correlates the number of teeth involved (quadrants) in the gingival resection with the percent of positive cultures. It is clearly shown that doubling the extent of surgery from 1 to 2 quadrants did not produce a significant increase in the percent of positive blood cultures found.

It has been suggested, in reports concerning the incidence of bacteremia after oral surgery,[8] that extractions from the upper arch are more prone to produce bacteremia than extractions from the lower arch. This has been linked to the number of roots involved. However, Table 5 shows the result of such a comparison on the basis of quadrants. A variation in the per cent of positive blood cultures was evident when upper and lower quadrants were considered independently, the lower quadrant giving a higher percentage. However, when the data was submitted to a Chi square analysis it showed no significance (Table 5). Several factors could influence such findings between quadrants. For example, the type of anesthesia was different. Infiltration anesthesia was used in upper quadrants, while in the lower, a mandibular block was used. Also, the position of the mandible may favor drainage through the vessels by simple gravity. The blood and the saliva tend to stagnate in the lower jaw, thus providing a greater opportunity for microorganisms to invade the vessels. Table 6 shows that *Streptococcus viridans* is the organism most frequently found in this study regardless of the source. However, the mere occurrence of an organism in the sulcus or on the gingival surface did not determine the frequency of findings in the blood cultures. For example, *Neisseria catarrhalis* occurred as the second most common organism in the sulcus and on the gingival surface, but it was not found in any of the blood cultures. In a previous study Cooley and Haberman[9] did find occasional members of the Neisseria group in blood cultures following extractions.

The group of 25 patients from whom blood samples were collected during surgery performed on different dates showed similar results in both tests. Seventeen of these patients gave negative cultures in both instances. Six patients had positive blood cultures twice and only 2 showed growth in the first sample but not in the second. Among the six patients having positive blood cultures on both occasions, only two showed the same organism while the remaining 4 yielded a different organism from each test. Similar results were obtained by Kraus, et al.[15] who reported 13 patients developed bacteremia during each of 2 to 4 operations. In their study, only

TABLE V

Comparison of incidence of bacteremia with quadrants involved in the operation

Quadrant Involved	*No. of Cultures*	*Avg. Depth of Gingival Sulcus over 3 mm.*	*Average Mobility of Teeth*	*No. of Positive Cultures*
Upper	26	3.81	1.13	3 (11.54%)[a]
Lower	19	3.76	1.12	6 (31.58%)[a]
Upper & Lower	69	...[b]	...[b]	19 (27.54%)
Total	114	....	....	..

[a]P=>0.10 for uppers vs. lowers.

[b]Items not done.

Chi square calculations for categories uppers and lowers.

	Positive	*Negative*	*Total*	*% Positive*
Uppers	3	23	26	11.54
Lowers	6	13	19	31.58
Totals	9	36	45	20.04% Difference

$$X^2=\frac{45(39-138)^2}{(26)\ (19)\ (9)\ (36)}=2.4130$$

The demonstration of $X^2=2.4130$ with P=>0.10 indicates that the difference is not significant.

TABLE VI

Frequency of occurrence of various organisms in the various sites of culture

Type of Organism	*Gingival Sulcus Cultures*	*Gingival Surface Cultures*	*Blood Cultures*
Streptococcus viridans	113	111	15
Streptococcus hemolyticus	14	12	0
Streptococcus anhemolyticus	14	17	2
Diplococcus pneumoniae	39	43	2
Staphylococcus aureus	65	72	8
Staphylococcus albus	3	2	0
Neisseria catarrhalis	90	95	0
Neisseria sicca	33	40	0
Neisseria flava	23	27	0
Diphtheroids (Leptothrix?)	14	14	2
Yeast	14	18	0

TABLE VII

Sensitivity of the various microorganisms isolated from blood cultures to nine antibiotics

Organisms Isolated from Positive Blood Cultures	*Novobiocin*	*Tetracycline*	*Polymyxin B*	*Erythromycin*	*Oxytetracycline*	*Chlortetracycline*	*Chloromycetin*	*Dihydrostreptomycin*	*Penicillin*
Streptococcus viridans									
Case No. 2	H	H	R	H	H	H	H	R	H
3	H	H	R	H	H	H	H	M	H
4	H	H	R	H	H	H	H	M	H
10	H	H	R	H	H	H	H	H	H
12	H	H	R	H	H	H	H	H	H
19	H	H	R	H	H	H	H	M	H
30	H	H	R	H	H	H	H	H	H
60	H	H	R	H	H	H	H	M	H
61	H	H	R	H	H	H	H	M	H
70	H	H	R	H	H	H	H	M	H
78	H	H	R	H	H	H	H	M	H
83	H	H	R	H	H	H	H	M	H
89	H	H	R	H	H	H	H	M	H
97	H	H	R	H	H	H	H	M	H
99	H	H	R	H	H	H	H	M	H
Staphylococcus aureus									
Case No. 1	H	R	R	H	R	R	H	H	H
7	H	H	R	H	H	H	H	H	H
25	H	H	M	H	H	H	H	M	H
31	H	H	R	H	H	H	H	H	H
32	H	H	R	H	H	H	H	H	H
58	H	H	M	H	H	H	H	H	H
96	H	H	R	H	H	H	H	H	H
97	H	H	R	H	H	H	H	H	H
Diplococcus pneumoniae									
Case No. 38	H	H	R	H	H	H	H	H	H
52	H	H	R	H	H	H	H	M	H
Diphtheroids									
Case No. 56	H	H	R	H	H	H	H	H	H
68	H	H	H	H	H	H	H	H	H
Streptococcus anhemolyticus Case No. 59	H	H	R	H	H	H	H	M	H
87	H	H	R	H	H	H	H	M	H

3 of the 13 cases gave identical organisms on the repeated tests.

The microorganisms isolated from the blood cultures were studied as to their sensitivity to antibiotics. It was felt that this might be of importance in considering bacteremias due to dental surgery. It is well known that there is a great variance in the sensitivity of different species and strains to antibiotics. Also, there is no way, at present, of determining under what circumstances one might expect an invasion of the blood stream by bacteria of the oral cavity and consequently, there are no dependable prophylactic measures for the prevention of this complication. However, some benefit could be obtained if the antibiotic sensitivities of the organisms found in bacteremias which resulted from dental surgery were known. Such data could be used as a means of selecting an antibiotic of choice for the premedication of patients undergoing extensive dental surgery. For example, Table 7 shows such data for each of the positive blood culture cases obtained in this study. All organisms were highly sensitive to penicillin, chloromycetin, erythromycin and novobiocin. Case 1 yielded a strain of *Staphylococcus aureus* which was resistant to 4 of the antibiotics used. Cooley and Haberman[9] reported similar antibiotic studies on 40 organisms isolated from blood cultures collected after exodontia. In that series, only chloromycetin, aureomycin and terramycin

TABLE VII *Continued*

Interpretation of symbols used:

Antibiotics	*Concentrations in Paper Discs*		
Novobiocin	50 mcg	10 mcg	2 mcg
Tetracycline	30 mcg	10 mcg	5 mcg
Polymyxin B	30 mcg	10 mcg	5 mcg
Erythromycin	15 mcg	5 mcg	2 mcg
Oxytetracycline	30 mcg	10 mcg	5 mcg
Chlortetracycline	30 mcg	10 mcg	5 mcg
Chloromycetin	30 mcg	30 mcg	5 mcg
Dihydrostreptomycin	100 mcg	10 mcg	2 mcg
Penicillin	10 units	5 units	2 units

R= Resistant to all concentrations tested.
M= Resistant to lowest concentration, sensitive to the two higher amounts.
H= Sensitive to all concentrations tested.

showed high sensitivity to all of the organisms isolated. Thus, it is of importance to know which bacteria are sensitive to the various antibiotics that might be used in therapy or prevention.

In the field of periodontology it has been commonly accepted that bacteria do not invade the periodontal tissues, and that the pathological changes observed are brought about by bacterial enzymes and metabolic products. In this study, the serial microscopic sections prepared from the excised gingivae of 3 of the patients giving a positive blood culture and from 4 of those giving a negative blood culture were stained by the Brown and Brenn[5] modification of the Gram stain for differential staining of bacteria in tissue sections. Such preparations showed nidi of bacteria in the gingival tissues. Figure 1 shows rods, filaments and cocci that could be seen in areas near the gingival attachment. No difference could be established between the observations on tissue layers of the positive and negative blood culture cases.

Inasmuch as the gingival sulcus depth, mobility and number of teeth involved did not seem to influence the occurrence of bacteremias following gingivectomies, a possible explanation for the bacteremias may be found by the presence of these nidi in the epithelial and connective tissue layers. When an incision is made through these nidi of bacteria, the microorganisms could be disseminated throughout the incised surface and into the interstitial fluid, from where they are drained by the lymphatics into the blood stream or they may enter the severed blood vessels directly.

The determination of preoperative bacteremia was not undertaken in the patients involved in this study. However, the reports of other investigators have shown that preoperative bacteremia is an infrequent occurrence. Robinson, et al.[20] concluded from the fact that no streptococci could be recovered preoperatively from the blood of 270 patients that bacteremia does not occur spontaneously in presumably healthy individuals. From a similar survey, Cobe[7] was unable to detect any preoperative bacteremia in a group of 100 patients. In addition, his data revealed no statistical difference between patients with periodontal disease and the control group during a study of the effects of tooth brushing on the incidence of bacteremia. Unpublished data accumulated from the blood transfusion service of Baylor University Hospital[14] showed less than a 1% incidence of bacteria in the bloods collected from over 1,000 blood donors. On the other hand, O'Kell and Elliot[17] report 10.8% incidence of preoperative bacteremia on patients with periodontal disease, and furthermore, Elliot[10] later reported that the rocking of loose teeth was often sufficient to produce a bacteremia. The question as to the ease of producing a transitory bacteremia has

received considerable attention of late. Some workers are of the opinion that the mildest massage of infected gingivae, such as that induced by eating, often results in bacteremia. Such a unique view would place modern man in the singular position of having an almost continuous presence of bacteria in his circulatory system. If this were true, the continual onslaught of invaders would cause the reticuloendothelial system to operate at a fever-pitch of phagocytosis and antibody production to prevent the more serious consequences of miliary spread of infection to various organs of the body. The apparent lack of such sequellae in the population in general, and among people with gingivitis specifically, should serve as a deterrent to such a broadly applied and extreme view. There is little doubt from the above data and from the work of others, that bacteremia is not an uncommon event among patients having various dental procedures. However, a broad conclusion and an extreme application of such data to the general population is not justified.

SUMMARY

The incidence of 24.56% positive blood cultures from 114 samples obtained during gingival resection is comparable to data reported for exodontia. Attempts to determine which factors influenced the occurrence of these bacteremias revealed that tooth mobility and depth of gingival sulcus were not important. The greater extent of the surgery, as measured by quadrants showed a tendency to yield more positive blood cultures but this data was not significant by Chi square analysis. The time of collection of the blood culture with relation to the start of surgery was shown to be a minor factor in the frequency of detection of bacteremia.

The great number of positives occurred in the first 60 minutes when the most trauma was taking place.

The organism most frequently found in the blood cultures, gingival sulcus and on the gingival surface was *Streptococcus viridans*. The antibiotic sensitivity tests performed on the 29 organisms isolated from the blood cultures showed that all were highly sensitive to chloromycetin, penicillin, erythromycin, and novobiocin.

The microscopic sections (stained by Brown and Brenn method) showed nidi of microorganisms in the epithelial and connective tissue areas of the excised gingivae. The significance of this gingival tissue invasion is discussed.

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CONFERENCE ON JOURNALISM

An International Conference on Dental Journalism and Documentation will be held on Friday, September 11, 1959, at the New York Academy of Science, 2 East 63rd Street, New York City. It is being presented under the joint sponsorship of the Editorial Board and the Subcommittee on Documentation of the Federation Dentaire Internationale, the American Association of Dental Editors, and the Council on Journalism and Bureau of Library and indexing Service of the American Dental Association.

The Conference will be one of the official programs preceding the Centennial Session of the American Dental Association, September 14-18, and the 47th Annual Session of the Federation Dentaire Internationale, September 12-19, in New York.

The Conference will provide an excellent opportunity for an exchange of international views on the problems of dental journalism, documentation, indexing and abstracting. Contributors to the formal program include, at this time: Professors H. H. Stones, F. E. Lawton, G. L. Slack and A. E. W. Miles, England; Dr. Max Filderman, France; Prof. Gustav Korkhaus, Germany; Dr. Y. Kuttler, Mexico; Prof. K. Sato, Japan; Dr. M. Muruzabal, Argentina; Mrs. I. M. Robinson, United States, and Drs. R. H. Orr and Donald A. Washburn, United States. The Conference will conclude with a banquet Friday evening at the Manhattan Hotel to which all are invited.

Degree of Keratinization and Glycogen Content in the Uninflamed and Inflamed Gingiva and Alveolar Mucosa*

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IT IS common knowledge that full keratinization, parakeratosis, and absence of keratinization may occur in the normal gingiva;[1,2,3,4,5] furthermore that inflammation is always present in the crevicular region[6] and frequently involves other regions of the gingiva. Some recent studies of the free gingiva[7,8,9] have investigated the effects of inflammation on keratinization and glycogen deposition. They have in part led to contradictory findings, but have stressed the fact that variable degrees of keratinization may occur in the same specimen in different parts of the free gingiva.[1] The purpose of the present study is to compare the different regions of the free gingiva, the attached gingiva, and the alveolar mucosa of uninflamed specimens with respect to degree of keratinization and content of glycogen, further to reinvestigate the interrelations between inflammation, keratinization, and glycogen in these regions.

MATERIAL AND METHODS

This study is based on biopsy specimens in bilaterally symmetrical areas, in the cuspid region, taken from 52 men 27-70 years of age. The donors were patients in the V. A. Hospital at Hines, Illinois, who were about to be discharged from the hospital, but were undergoing denture work prior to returning home. The biopsies were taken after the extraction of the teeth. The case histories of the donors were carefully scrutinized for elimination of diabetes and debilitating diseases. Most of the men were cigarette smokers, some used other forms of tobacco, and a few were nonsmokers. No consistent difference between the groups could be discovered with respect to any of the traits studied. The biopsies were so taken as to include the alveolar mucosa. On histological examination the epithelium of the gingival pocket was rarely found to be complete. It was, however, possible in almost all cases to judge the severity of the inflammation in this region.

Immediately after removal, the specimens were fixed in 5% neutral formalin. Sections were cut at 5 μ thickness following paraffin embedding, and stained with hematoxylin-eosin, with Mallory's triple connective tissue stain or treated according to Hotchkiss' procedure for the demonstration of periodic acid-Schiff positive material (PAS).

Subdivision of gingiva. Separate estimates of inflammation, glycogen content, and degree of keratinization were made for the following regions: (1) region bordering the gingival sulcus [crevicular gingiva], (2) crest of the free gingiva [crest region], (3) oral surface of the free gingiva [oral surface], (4) attached gingiva, and (5) alveolar mucosa.

The boundaries between the first three regions were judged by their anatomical locations. The boundary between free and attached gingiva could usually be recognized from the location of the free gingival groove,[2] that between attached gingiva and the alveolar mucosa from the characteristically different histology of the alveolar mucosa.[10] In the occasional instances of doubt about the exact location of a boundary, only the central portions of the regions in question were included.

Degree of inflammation. The severity of inflammation was estimated from the density of the inflammatory cell infiltration in the connective tissue underlying a given epithelial region. Account was also taken of the number of inflammatory cells migrating through the epithelium and of the degree of edema in and between the epithelial cells. The degree of inflammation was estimated using a 6 point scale from 0 to 4+. The inflammatory cells usually consisted of lymphocytes and plasma cells; occasionally mast cells were seen.

Estimate of glycogen concentration. Glycogen was estimated by comparing the PAS-positive material in untreated sections stained with the PAS reagent and in control sections first exposed to the action of saliva and then stained with the PAS reagent. The difference in PAS positive material was considered as glycogen. Comparison of frozen-dried

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This investigation was supported (in part) by Research Grants D-658 and D-513 from the National Institute of Health, U. S. Public Health Service, and a Grant from the Tobacco Industry Research Committee.

TABLE 1.

Percent Incidence of 6 Degrees of Inflammation in Four Regions of Gingiva and in the Alveolar Mucosa.

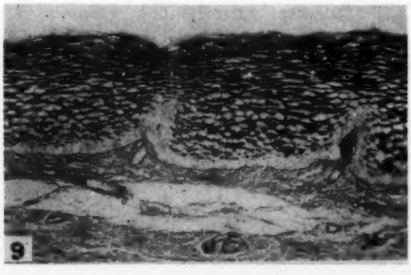
Degree of inflammation	*Crevicular region*	*Crest*	*Oral Surface*	*Attached Gingiva*	*Alveolar Mucosa*
−	0	34	56	40	51
±	10	4	8	19	24
+	11	38	30	25	21
++	34	13	5	13	3
+++	41	12	1	4	1
++++	3	0	0	0	0
Total No. of Cases	70	77	79	85	71

sections with formalin fixed sections showed no significant loss of glycogen in the epithelial cells due to formalin fixation, although the intracellular distribution was altered.

The amount of glycogen present was estimated on a 6 point scale, ranging from zero to 4+. In the gingiva, the basal cells, the adjacent two rows of prickle cells, and the most superficial cell layers were free of glycogen in all specimens. In the alveolar mucosa the deepest cell layers were free of glycogen, as in the gingiva, but glycogen was contained in the most superficial cells (fig. 9 and 10). In the gingiva, glycogen might occur in larger or smaller amounts in all or nearly all of the intermediary cell layers of a given region, or might be confined to a few layers or small islands within a region. The ratings took account of the amount per cell and of the proportion of cells containing glycogen. The amount of glycogen in regions of the gingiva was often considerable, though it did not reach the highest of the concentrations seen in the alveolar mucosa.

Independent ratings of inflammation and of glycogen content made by two observers usually coincided, or were at most apart by one point. The subdivision of the gingiva into four separate regions facilitated this semiquantitative approach.

Degree of keratinization. Mallory's triple connective tissue stain, using the concentrations and timing specified by Bensley and Bensley,[11] and hematoxylin-eosin staining were used for evaluating keratinization. In Mallory stain, *fully keratinized* (fig. 2) and *parakeratotic layers* (fig. 4) have a bright red color, the underlying layers being blue. The staining of the nuclei in parakeratotic layers is indistinct so that the distinction of full keratinization and parakeratinization requires comparison with H-E stained sections. *Incompletely parakeratotic regions* (fig. 6) have a blue sur-



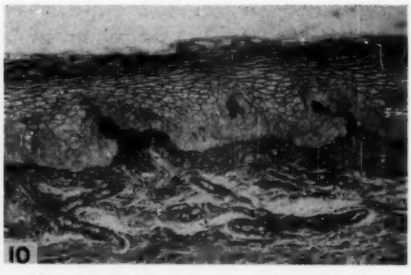


Fig. 9 and 10: Photomicrographs of a region of alveolar mucosa. Fixation 5% neutral formalin. Mag. X86.
9—Section treated with the periodic acid-Schiff reagent.
10—Section from the same block incubated with diastase preceding PAS treatment.
Note the cytoplasmic staining in section 9 and its absence in the control section 10. Only the cellular membranes remain PAS-positive after diastase treatment.
Note in section 9 the absence of glycogen in the basal and adjacent Malpighian layers, the presence of glycogen in the most superficial layers of epithelium, and its localization in the basal aspects of the cells, which is characteristic for formalin-fixed tissue. The section illustrates rating 3+ for glycogen concentration.

face edge, a red-staining layer underneath the surface edge, and blue-staining cellular layers basal to the red layer. The nuclei are recognizable in the most superficial layers as in parakeratosis. In *nonkeratinized epithelium* (fig. 8) all layers stain blue.

FINDINGS

Incidence of inflammation. The highest incidence of inflammation was found in the crevicular region. This region showed some degree of inflammation in all specimens and moderate or severe inflammation in 80%. Next in incidence and severity was the adjacent region at the crest of the gingiva. The least inflamed region was the oral surface of the free gingiva, and similar to it was the alveolar mucosa. The attached gingiva showed only slightly less inflammation than the crest region. The differences between these four regions were small, however, when compared with the much higher degree and incidence of inflammation in the crevicular region (Table 1).

Spread of inflammation from crevicular region to remainder of gingiva and alveolar mucosa. The degree of inflammation at the crevicular region was always found to be more severe than in any other region of the same specimen.

In Table 2 the specimens are grouped according to whether inflammation in the crevicular region was mild, moderate, or severe. Column 2 shows the percentage of cases in which the average inflammation in the other four regions of the specimen was moderate or severe and column 3 shows the percentage of regions which were entirely free of inflammation.

In the group with mild inflammation in the crevicular region, the entire remainder of the tissue showed at most mild inflammation, and 73% of all regions were entirely free of inflammation. When inflammation in the crevicular region was moderate, 14% of the specimens averaged more than slight inflammation in the remainder of the gingiva. The percentage of regions which had no inflammation had dropped to 44. In the group with severe inflammation in the crevicular region, the other regions of the gingiva were severely or moderately inflamed in 59% of the specimens. The percentage of regions which were free of inflammation had dropped to 26.

TABLE 2.

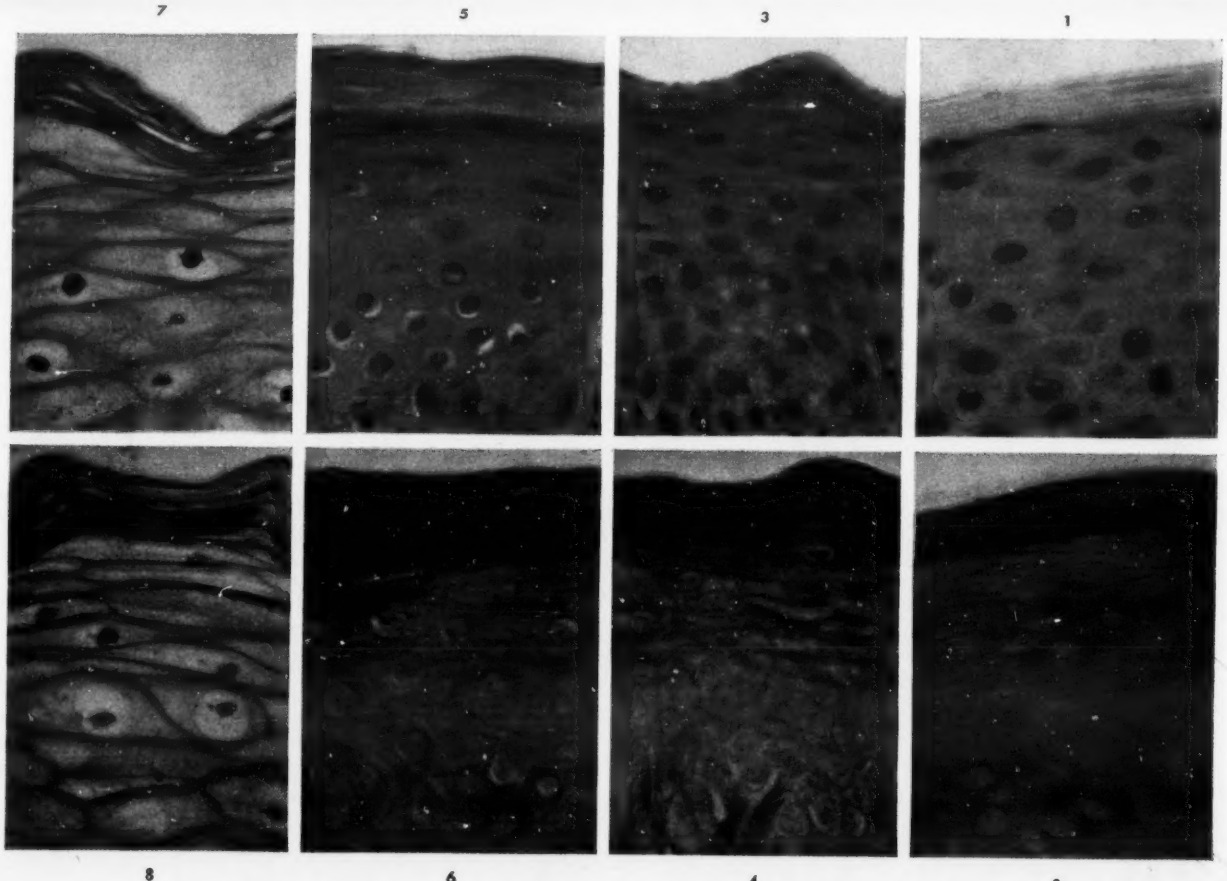
Relation of inflammation in crevicular region to inflammation in other gingival regions of the same specimen.

Degree of inflammation in crevicular region	*Percentage of specimens averaging moderate or severe inflammation in other regions*	*Percentage of other regions which were free of inflammation*
+ and ±	0	73
++	14	44
++++ and +++	59	26

This table includes only the 62 specimens in which the degree of inflammation could be evaluated in all 5 regions.

The sharp decrease in the percentage of uninflamed regions and the sharp increase in the average severity of inflammation showed that the presence and degree of inflammation in the entire gingiva depended on the degree of inflammation at the crevicular region. The strength of this influence decreased progressively with the distance from the crevicular region, leading to decreasing severity of inflammation in going from the dental surface of the free gingiva to the alveolar mucosa.

Comparison of inflammation in bilateral pairs of corresponding regions of gingiva. Since specimens from the corresponding opposite regions of the oral cavity were available for most of the biopsies, it was possible to compare the inflammation in the two specimens from the same patient. Statistical analysis showed that the severity of inflammation on the right was independent of the degree of inflammation on the left. The severity seems to depend mainly on local causes, and it is unlikely



FIGS. 1–8: Microphotographs of human gingiva and alveolar mucosa. Mag. × 550. FIGS. 1, 3, 5, 7, H–E; Figs. 2, 4, 6, 8 Mallory staining.
FIGS. 1 AND 2: Region showing full keratinization.
FIGS. 3 AND 4: Region showing parakeratosis.
FIGS. 5 AND 6: Region showing incomplete parakeratosis.
FIGS. 7 AND 8: Region showing nonkeratinization.

TABLE 3.
Percent incidence of four degrees of keratinization in noninflamed regions of gingiva.

	Crest	*Oral Surface*	*Attached Gingiva*
Keratinization	18	22	32
Parakeratosis	27	67	68
Incomplete Parakeratosis	36	11	0
Nonkeratinization	18	0	0

that in the material included in this study a systemic factor played a significant role in determining the degree of inflammation.

UNINFLAMED GINGIVA

Incidence of different types of keratinization in uninflamed regions of gingiva (Table 3). A tendency to higher degrees of keratinization was noted in going from the crest to the oral surface of the free gingiva and to the attached gingiva. *At the crest* all four degrees of keratinization were observed, most frequently incomplete parakeratosis, next parakeratosis. Full keratinization occured in 18%. At the oral surface of the free gingiva only three degrees of keratinization were noticed. No specimen showed nonkeratinized epithelium. Incomplete parakeratosis was less frequent than at the crest, and the most frequent type was parakeratosis. Full keratinization occurred in 22%. In the attached gingiva only two types of keratinization occurred; there were no cases of incomplete parakeratosis and no cases of nonkeratosis. Here too, the majority of cases were parakeratotic, but full keratinization increased to 32%.

The epithelium of the alveolar mucosa was found nonkeratinized in all instances. The boundary between the keratinized or parakeratinized epithelium of the attached gingiva and the nonkeratinized epithelium of the alveolar mucosa was sometimes sharp, sometimes a transitional zone of incomplete parakeratosis was seen.

Comparison of the degree of keratinization in the two specimens from the same patient showed a tendency to bilateral symmetry. This tendency was found in all three regions and all four kinds of keratinization, but was not marked enough for a statistically significant deviation from chance expectation.

Glycogen content of the uninflamed gingiva (Table 4). Glycogen occurred in all three regions of the gingiva, but not in all specimens and not in the same high concentration as in the alveolar mucosa.

The three regions of gingiva differed progressively in incidence and concentration of glycogen. At the crest, glycogen occurred most often and in the highest concentration, only one third of the specimens containing no glycogen. In the attached gingiva, glycogen occurred least often and in the lowest concentrations, half the specimens containing no glycogen in this region. The oral surface of the gingiva was intermediary in incidence and concentrations.

Glycogen occurrence in the uninflamed gingiva therefore also showed a regional gradient, but in the opposite direction as that of the tendency for keratinization.

All specimens contained glycogen in the epithelium of the alveolar mucosa, the large majority in higher concentrations than were encountered in the gingiva. The inverse relation between glycogen and keratinization, which in the three regions of gingiva was noted as opposite gradients of incidence, was absolute in the alveolar mu-

TABLE 4.

Percent incidence of six glycogen ratings in uninflamed regions of gingiva and alveolar mucosa.

Glycogen Rating	*Crest*	*Oral Surface*	*Attached Gingiva*	*Alveolar Mucosa*
−	34	46	50	0
±	8	10	29	6
+	23	32	8	11
++	31	12	13	31
+++	4	0	0	44
++++	0	0	0	8

TABLE 5.

Percent incidence of four degrees of keratinization in inflamed and uninflamed gingival regions.

	Crest			*Oral Surface*			*Attached Gingiva*		
	Inflamed		*Un-*	*Inflamed*		*Un-*	*Inflamed*		*Un-*
	Severe	*Mild*	*inflamed*	*Severe*	*Mild*	*inflamed*	*Severe*	*Mild*	*inflamed*
Keratinization	6	4	18	0	6	22	0	8	32
Parakeratosis	6	18	27	80	58	68	64	86	68
Incomplete parakeratosis	47	57	36	20	36	11	29	3	0
Nonkeratinization	41	21	18	0	0	0	7	3	0

cosa, all specimens having glycogen and no specimen being keratinized to any degree in this region.

The two bilateral specimens showed a tendency for similar glycogen concentration. This was particularly marked for the cases in which glycogen was absent.

INFLAMED REGIONS OF GINGIVA

Effect of inflammation on keratinization (Table 5). Inflammation depressed the keratinization of the three gingival regions. The magnitude of the depressing effect was found to be related to the level of keratinization characteristic for the uninflamed epithelia of each region and to the severity of the inflammatory process.

At the crest, the incidence of keratosis and parakeratosis was reduced below the normal low percentage, and incomplete parakeratosis and nonkeratinization occurred in 80% of the cases. In severe inflammation the latter two amounted to 90% of the cases.

At the oral surface, the number of cases showing full keratinization decreased in mild inflammation. None were found in severe inflammation. Instead, the number of cases showing parakeratosis and incomplete parakeratosis increased. As in uninflamed specimens, however, there were no cases of nonkeratinization.

In the attached gingiva, mild inflammation sharply decreased the incidence of full keratinization and raised that of parakeratosis. In severe inflammation, full keratinization disappeared and parakeratosis decreased; instead, incomplete parakeratosis and nonkeratinization appeared.

Effect of inflammation on glycogen content. The presence of inflammation increased the incidence and concentration of glycogen in all three areas of gingiva. Mild inflammation reduced the incidence of glycogen-free epithelia from 34% to 13% in the crest region, from 46% to 27% at the oral surface, and from 50% to 41% in the attached gingiva, raising instead the incidence of low and of moderate concentrations of glycogen. In the presence of severe inflammation, there were almost no regions without glycogen. The large majority of cases showed considerable amounts both at the crest and the oral surface. The attached gingiva was somewhat more resistant to glycogen deposition, half the specimens still having only small amounts.

By contrast to the marked effect inflammation had on the glycogen content of the gingiva, its presence or absence made almost no difference to the glycogen content in the alveolar mucosa. However, in the few specimens in which the alveolar mucosa was severely inflamed, the glycogen content was maximal. Conversely, in the few specimens in which the alveolar mucosa contained little glycogen, there was no inflammation.

TABLE 6.

Percent incidence of four glycogen ratings in inflamed and uninflamed gingival regions.

Degree of inflammation	Crest			Oral Surface			Attached Gingiva			Alveolar Mucosa		
	Severe	Mild	None	Severe	Mild	None	Severe	Mild	None	Severe	Mild	None
Glycogen rating:												
−	21	13	34	0	27	46	14	41	50	0	0	0
±,+	5	48	31	0	37	42	50	43	37	0	17	17
++,+++	74	39	35	100	37	12	36	14	13	0	67	75
++++	0	0	0	0	0	0	0	0	0	100	17	8

DIFFERENCES BETWEEN ADJACENT REGIONS

Crevicular epithelium and crest region. In 15 specimens, the degree of inflammation was the same in the crevicular and the crest region. In 52 specimens, the two regions differed, the crevicular region being more severely inflamed in all of the 52 specimens.

Crest region and oral surface of free gingiva. In the majority of specimens, the crest region differed from the adjacent region of the free gingiva with respect to inflammation, keratinization and glycogen content. Inflammation was of different severity in 41 specimens, alike in 35. The degree of keratinization was different in 52 specimens, identical in 21. The glycogen content differed in 43 and was alike in 32 specimens. In the specimens in which these two regions differed, the crest region was always the more severely inflamed. It had usually the lower degree of keratinization and the higher concentration of glycogen.

Oral surface of free gingiva and attached gingiva. As was shown in the preceding sections, the percentage incidence figures for the different degrees of inflammation, keratinization and glycogen content were rather similar for the oral surface of the free gingiva and the attached gingiva, which might lead one to suppose that in most specimens there would be no difference between the two regions. However, this was not the case. It was more common to find differences in the ratings for the two regions than to find them alike. Different degrees of inflammation were seen in 45 specimens, different degrees of keratinization in 27, and different glycogen content in 44 specimens. In the differing specimens, it was sometimes the oral surface, and sometimes the attached gingiva which had the higher degree of inflammation, the higher degree of keratinization, or the higher content of glycogen.

Attached gingiva and alveolar mucosa. Inflammation was found to be of identical degree in attached gingiva and alveolar mucosa in 34 specimens and to differ in 40. In the differing specimens, commonly the attached gingiva was more severely inflamed. The degree of keratinization was always different except in the few specimens where the epithelium of the inflamed attached gingiva was not keratinized. Conversely, the glycogen content in the alveolar mucosa was higher than that of attached gingiva in all but three specimens, in which the alveolar mucosa contained unusually small amounts of glycogen.

DISCUSSION

1. *Characteristics of the uninflamed gingiva. Keratinization.*

Clinically normal and histologically uninflamed regions of gingiva show four degrees of keratinization with regionally different incidence figures. Full keratinization is least frequent in the crest region and most frequent in the attached gingiva. Parakeratosis is more frequent than full keratinization in all regions of gingiva,

accounting for a fourth of the specimens at the crest and two-thirds of the oral surface and of the attached gingiva. Incomplete parakeratosis is the largest single class at the crest; it is infrequent at the oral surface and absent in the attached gingiva. Absence of keratinization occurs only in the crest region, where it accounts for a fifth of the cases.

The common occurrence of parakeratosis has not yet found its way into the text books. However, the present study confirms a number of more recent investigations in this respect.[12,13,7] A comparison of the figures given in these reports with our incidence figures does not seem warranted because of differences in the age distribution, differences in the criteria for diagnosing the presence of inflammation, and the separation of incomplete parakeratosis from parakeratosis, which is new to the present study.

Regional gradient in tendency for keratinization. The differences in the regional incidence of the four degrees of keratinization add up to a regional gradient of increasing keratinization in going from the crest to the attached gingiva. This can be seen from the successive increase in incidence of the two more advanced degrees of keratinization and the corresponding stepwise decline in the incidence of the less advanced degrees. From the studies of Dewar[9] and Trott[7] it appears that the crevicular epithelium is unkeratinized even in the rare cases when this region is virtually free of inflammation. If this should be confirmed, the gradient may be said to begin with the absence of keratinization in the crevicular region of the free gingiva and to progress from there to higher degrees of keratinization over the crest and oral region of the free gingiva to the attached gingiva.

Glycogen content. The epithelium of uninflamed regions of gingiva contained glycogen in more than half of the specimens but rarely in large amounts. This finding is in agreement with that of Trott,[8] but disagrees with the findings of Dewar,[9] who concludes that glycogen is in all cases associated at least with traces of inflammation. Applying the same rigorous standards as this author, we still find glycogen in cases in which all signs of inflammation are lacking. Analysis of such cases has shown[10] that they tend to occur in individuals in whom a bilateral high concentration of glycogen is found in the alveolar mucosa. These individuals may have a systemic predisposition for the accumulation of glycogen.

Regional gradient in the incidence of glycogen and inverse relation of glycogen and keratinization. The incidence and concentration of glycogen were highest in the crest region, intermediary at the oral surface and lowest in the attached gingiva. This gradient goes in the opposite direction of that of the gradient in the tendency for keratinization. The antagonistic relation between keratinization and glycogen deposition which is indicated by these opposing gradients has been noted in other stratifying epithelia.[14,15,16] Among the regions included in the present study this antagonism is most marked in the alveolar mucosa, which was not keratinized and contained glycogen in all specimens in relatively large amounts.

2. *The effect of inflammation on keratinization and glycogen content of the gingiva.* Inflammation was found to depress the degree of keratinization in proportion to its severity, and to raise incidence and concentration of glycogen, also in proportion to its severity. The latter finding is in agreement with the findings of both Trott and Dewar, and also agrees with observations in other epithelia.[17,18,19] The crest region, even when it is not inflamed, tends to be less keratinized and to contain more glycogen than the oral surface of the free gingiva. In addition, it is more often and more severely inflamed than the oral surface and then of even lower keratinization and higher glycogen content. The regional gradients in keratinization and glycogen content therefore are steeper

when uninflamed and inflamed regions are considered together than for the uninflamed specimens alone. In other words, this gradient is more marked when the different regions are compared without regard to inflammation.

3. *Limitations of the present study.* Because even clinically normal gingiva is so frequently found inflamed on microscopic examination, most investigations of the "normal" gingiva are confined to a limited number of cases and therefore to tentative conclusions as regards incidence figures for degrees of keratinization and glycogen content in an uninflamed gingiva. The present study is no exception in this respect. It is, however, based on larger biopsy specimens than are generally available. Most of the specimens included the three regions of the free gingiva, attached gingiva and alveolar mucosa in the same section. For this reason, it was possible to compare the five regions under identical staining conditions and therefore to draw at least tentative conclusions about the inherent differences between different regions of the gingiva. A further advance beyond previous studies is seen in the introduction of incomplete parakeratosis as a fourth type of keratinization. Evidence for the identification of this type as intermediate between parakeratosis and nonkeratinization was presented in a separate report.[20] The separation of incomplete parakeratosis from parakeratosis improves the statistical correlation between degree of keratinization and the severity of inflammation and also between degree of keratinization and glycogen content, because change to incomplete parakeratosis appears to be the first reaction of a parakeratotic epithelium to mild inflammation or to small amounts of glycogen. When such specimens are classified as parakeratotic, it appears as though the presence of mild inflammation and of small amounts of glycogen had no effect on the degree of keratinization.

SUMMARY

Degree of inflammation, keratinization and glycogen content were studied in bilateral biopsies of gingiva and alveolar mucosa obtained from 52 men 27-70 years of age. Four types of keratinization and variable amounts of glycogen were observed in uninflamed as well as in inflamed gingiva. These traits were evaluated separately at the crest of the free gingiva, the oral surface of the free gingiva, the attached gingiva, and the alveolar mucosa.

1. In the absence of inflammation it was found that the tendency for keratinization shows a regional gradient, being least marked in the crest region and most marked in the attached gingiva.

Likewise, a regional gradient in incidence and concentration of glycogen was noted, the deposition being most marked at the crest and least marked in the attached gingiva.

2. Study of inflamed regions showed that inflammation reduces the tendency for keratinization and increases the tendency for deposition of glycogen.

3. The alveolar mucosa has a nonkeratinizing epithelium and contains glycogen in all specimens.

4. Keratinization and glycogen content are inversely related in uninflamed gingival regions, more markedly in inflamed gingival regions and most markedly in the alveolar mucosa.

ACKNOWLEDGMENTS

The help of Dr. Harry Sicher in critical reading of the manuscript, of Dr. M. Massler for his interest and cooperation, and of Mr. W. Winn, biological photographer, in preparing the microphotographs, further of Dr. N. Kanof, editor of the Journal of Investigative Dermatology, for permission to reproduce figs. 1-8 is gratefully acknowledged.

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38th ANNUAL MEETING OF A.A.W.D.

A meeting of the Association of American Women Dentists will be held in the Hotel San Moritz, 59th Street and Central Park South, New York City, on Monday, September 14, 1959.

A breakfast and business meeting are called for 9 A.M., and a banquet at 6:30 P.M.

A hospitality room will be maintained at the San Moritz, to which visiting women dentists will be welcome.

For further information, address Dr. Goldanna Cramer, Secretary, 95 West Broadway, Salem, N. J.

Lillian Barkann, D.D.S.,
Chairman, Publicity Committee

A Study of the Gingival and Periodontal Conditions of a Group of People in Chieng Mai Province†

by M. R. Nibhasara Ladavalya, D.D.S.‡ and Robert Harris, M.D.S. (Syd.),*
Sydney, Australia

IN addition to the study of the dental caries experience of Thai children, which is the subject of another report, the status of their gingival tissues was also noted. Other groups were examined, including a kindergarten school in Chieng Mai with a total enrolment of 250 boys and girls. Of these latter children in the age groups 4 through to 9 years, 187 were examined and the presence of gingivitis and enamel hypoplasia noted. A number of older patients, including student nurses at Chieng Mai Hospital, were also examined. The presence of gingivitis was recorded if the color of the tissues were darker than the normal pink and the contours of the gingival crest and papillae were enlarged. Examination for the extent of pocket formation in the older patients was made by blunt probe; the site selected was the upper and lower central and the upper and lower first molar regions.

In the case of the student nurses it was possible to examine their teeth for dental caries, but X-rays were not taken.

RESULTS

A. Chieng Mai and Sarapi Primary School Children.

Gingivitis

In the eighty children between 9 and 16 years, the gingival condition is recorded in Table 1.

Table I

Status of gingival tissues in 80 children 9—16 years (Chieng Mai and Sarapi)

Gingival Status	*Boys*	*Girls*	*Total*
Normal gingiva	2	8	10
Mild gingivitis	12	12	24
Moderate gingivitis	3	12	15
Severe gingivitis with hyperplasia	21	10	31
TOTAL	38	42	80

Periodontal pockets present in 3 of the severe cases.

Calculus

In the eighty children between the ages of 9 and 16 years, calculus was present in sixty and the distribution according to zones in the dental arches is shown in Table 2.

B. Chieng Mai Kindergarten.

The age range in this school covered the years 4-9 and 187 children were available for examination.

In addition to the presence of gingivitis the extent of enamel hypoplasia was recorded.

The results are recorded in Table 3.

This shows 25.1 per cent of children with gingivitis and 20.3 per cent with enamel hypoplasia.

C. Buagkaing Village School.

At a village center situated in a farm-

Table II

Location of calculus in 33 boys, 28 girls 9—16 years. Chieng Mai and Sarapi.

Region	*No. of Children*
Anterior	30
Posterior	18
All quadrants	13
TOTAL	61

†Survey carried out in 1958 when one of the authors (R.H.) was serving as a WHO consultant in dental health to the Government of Thailand.

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TABLE III

Presence of gingivitis and enamel hypoplasia in 187 (94 girls, 93 boys) Chieng Mai Kindergarten children aged 4—9 years.

Age	*Boys*	*Gingivitis*	*Enamel Hypoplasia*	*Girls*	*Gingivitis*	*Enamel Hypoplasia*
4	15	—	1	8	—	1
5	21	—	6	13	5	4
6	14	4	3	33	7	8
7	20	5	4	17	8	4
8	16	7	2	15	6	3
9	7	3	1	8	3	1
TOTAL	93	19	17	94	29	21

ing district about 5-6 kilometers along a track from Sankampang, the opportunity was taken to visit a small school and a class of eighteen boys and twelve girls in the age group 8-10 years was examined. All the children in this group had gingivitis, half in a severe form. Oral hygiene was poor and heavy deposits of calculus were present in all mouths.

D. Health Center Patients at Sankampang.

This center is situated about fifteen kilometers by road from Chieng Mai and is served by nurses who carry out instructions of the medical officer who attends once each week. Health education is given in prenatal programs. At the time of this visit, 32 patients were in attendance for the morning session; sixteen of these were children under the age of seven years and seven of these were caries free. Gingivitis was not evident, although oral hygiene was not perfect. Most of the children were chewing glutinous rice. All children had enamel hypoplasia.

The adult patients were all women, none being older than forty years. Some indulged in betal-nut chewing, and, in all, oral hygiene was poor and gingivitis was present. Eleven had periodontal disease, manifested by mobility of teeth and recession of gingivae; one, five months' pregnant was edentulous, and, as might be anticipated, did not have dentures. Vitamin B group deficiencies were noted by the presence of oral lesions (glossitis and angular cheilosis) in five of the oldest women.

A summary of the extent of gingivitis in the children is shown by Table 4.

E. Student nurses at Chieng Mai Hospital.

The 37 student nurses have a secondary education of the School Certificate Standard and their ages range 18-29 years.

Gingivitis was present in 30 (severe in nine and very severe in one). Calculus was present in 29 (heavy deposits in nine and very heavy in one). Pocket formation was moderate (3 mm) in nine and severe (6

TABLE IV

Distribution of children with gingivitis

	Number of Children Total		*Gingivitis*	
	Girls	*Boys*	*Girls*	*Boys*
Chieng Mai and Sarapi School	40	40	32	38
Chieng Mai Kindergarten	94	93	29	19
Buagkaing	12	18	12	18
Sankampang Health Centre	10	6	—	—
	156	157	73	75

TABLE V

Distribution of adults with gingivitis.

	Number of adults		*Gingivitis*	
	Female	*Male*	*Female*	*Male*
Chieng Mai Hospital Patients	10	24	9	21
Chieng Mai Hospital Nurses	37	—	30	—
Sankampang	16	—	15	—
	63	24	54	21

mm) in one. Attrition was noted in eight and in five of these the condition was advanced.

The caries experience expressed as D.M.F. teeth, was 112, an average rate per person of 3.0. Of this figure of 112 affected teeth, 73 were decayed, 38 were missing and one was filled.

Fifteen mouths were caries free and four of these had marked attrition.

F. Adult patients in Chieng Mai Hospital.

These were adults in the age range 39-66 years and included three Buddhist Priests; approximately half were in-patients. Three indulged in the practice of betel-nut chewing and eleven had a full complement of teeth.

Of the 24 male patients, age range 39-66 years, three were free of calculus and gingivitis, 13 had some degree of attrition (10 severe, including two free of gingivitis and six with deposits of calculus and gingivitis) 6 without calculus and 18 with moderate (3 mm) to severe (6mm) pocket formation and mobility of teeth.**

Of the 10 female patients range 42-56 years, one patient was without gingivitis or calculus deposits, eight had moderate to severe pocket formation and mobility of teeth and four had some degree of attrition.

A summary of the extent of the gingivitis in adults is shown in Table 5.

DISCUSSION

It will be noted that a total of 313 children was examined and 148 (73 boys, 75 girls) had some degree of gingivitis. Calculus was present in the mouths of 91 (51 boys, 40 girls).

In the older age groups, 24 males and 63 females, were examined. Gingivitis was present in 65 (21 males, 54 females). Calculus was present in the mouths of 75 (18 males, 57 females). In the children it is noted that the lower anterior region was the site of greatest prevalence of calculus, followed by the upper posteriors and then all quadrants. In the older patients, including the student nurses, all quadrants were involved and some patients had large amounts of calculus.

In all groups studied where the patient had normal gingiva, some form of oral hygiene was practiced. For example, in the school children, two girls with excellent mouths used a dentrifice and toothbrush twice daily. In the older patients, five of the student nurses had normal gingiva, but one only of these was caries free, and in the adults a sixty-five year old man had an excellent dentition and normal gingiva and two Buddhist Priests 39 and 65 years of age had excellent gingival tissues. The latter two had been in the habit of using a dentifrice and toothbrush regularly and the other man had always rinsed his mouth with water after a meal, and did use a toothbrush, but not regularly. It should be noted that it is a strict custom of the

**Time did not permit a detailed survey and recording of each tooth, but where teeth from any of the upper and lower first molars or upper and lower central regions were present, a measurement of the depth of the gingival sulcus was made and noted.

Buddhist Priest to have only two meals daily and these must be taken before noon.

Gingivitis becomes evident at the 6-7 year age group and approximately 45.7 per cent of all the children examined had gingivitis. Oral hygiene is in most cases absent and calculus was found in the mouths of 29 per cent (91) of these children.

In the intermediate group represented by the student nurses, 81 per cent (30) had gingivitis and 78.3 per cent (29) had calculus, attrition was present in 18.9 per cent (7) of these mouths.

Periodontal pocket formation was noted in 50 per cent (36) of the older age groups. Student nurses—10. Adult patients—26.

In the older patients examined 98.2 per cent (30) had calculus. Attrition was present in 33 per cent (11) of these mouths.

In each mouth where oral hygiene was practiced the gingival tissues were normal.

Occlusion based on contact of opposing teeth and functional movements rather than according to Angle's Classification shows that in the student nurses and older patients where attrition is marked, the patients had fair occlusion, but did not necessarily possess a full complement of teeth. Of the 61 patients, 45 had 30 or more teeth and of these 18 had marked attrition.

SUMMARY AND CONCLUSION

The mouths of a number of patients covering all ages were examined for the presence of gingivitis, calculus, periodontal pocket formation and attrition. The mouths with good oral hygiene were free from disturbances in all age groups. The standard of oral hygiene would appear to be a factor in the causation of gingival and periodontal lesions, and the prevalence of enamel hypoplasia in the deciduous dentition should be noted. Other factors, particularly nutrition, need to be considered.

There is a need for a careful survey of the prevalence and a study of the causes of gingival and periodontal lesions in this region.

ACKNOWLEDGMENT

It is desired to express an appreciation to Dr. Rabieb Rerkasem, Director, and to Dr. Thawon A. Rajathon, dentist, Nakorn Chieng Mai Hospital, without whose co-operation and assistance this survey could not have been undertaken and to Professor See Sirisinha, Dean, School of Dentistry, Bangkok, Thailand, who undertook the organization of this journey.

Robert C. McKay, 115 East 61st Street, New York, New York, reports that he has found it helpful in his attempts to convince patients of the importance of home care, to give them the following quotation:

"Emphasis should be placed by the dentist on the value of home care in the maintenance of good oral hygiene. Unless the patient is motivated to maintain good oral hygiene carefully, tediously and persistently, the dentist's treatment of periodontitis, no matter how complex, will result in temporary improvement only." (Hine, M. K. Preventive Periodontics. J.A.D.A., Vol. 55, (Nov.) 1957. pp. 668-673.)

Case Report . . . .

Leukemia Involving the Oral Cavity

by IRWIN WALTER SCOPP, B.S., D.D.S.* AND ARTHUR M. QUART, B.A., D.D.S.,**
New York, N.Y.

THE dentist may at times have the responsibility for the early diagnosis of leukemia. Patients in all stages of this disease have oral symptoms, and the dentist may be the first professional man consulted by leukemic patients for gingival bleeding, enlargement of the gingiva, toothache, ulcerative lesions of the gingiva or petechia in or about oral cavity.

Fortunately, patients with leukemia do not frequently walk into the dentist's office for therapy. It has been estimated, however, that approximately 5,000 deaths in the United States alone may occur from this disease annually. This is more deaths than occur from poliomyelitis, malaria, typhoid fever, small pox, and diphtheria combined. Obviously, a dentist who treats the swollen and bleeding gums or extracts a tooth of a leukemic patient is in for severe criticism. Familiarity with the oral symptoms of this disease assists in possible early diagnosis and permits more effective suppressive therapy.

CASE REPORT

This is the second admission for a 68 year old, white, unmarried male to the New York Veterans Administration Hospital, New York 10, N. Y. and the date of this admission was November 18, 1957. His chief complaints were bouts of weakness (which have occurred with increased frequency since July, 1957), shortness of breath, "lumps" in both axilla and neck, ecchymosis on dorsum of left hand, and neck pains.

History of Present Illness: In July 1956 patient developed "weak spells" and with minimal exertion these spells were accentuated. He also developed some dyspnea which he noted especially when smoking. His condition at that time was thought to be arteriosclerotic heart disease, and several EKGs were taken but they were essentially normal. Despite good appetite, patient lost 20 pounds since July 1956. His "weak spells" have been getting more frequent and more severe since that date.

The patient's illness became acute in May-June, 1957 when he bled excessively on three different occasions following tooth extractions by his private dentist. Bleeding episodes started two to three days after extraction and at times were severe. There was no history of oral bleeding or ecchymosis prior to extractions. Patient had teeth extracted one at a time spaced three weeks apart. Before third extraction, vitamins were given but bleeding was as severe as on prior extractions. Following the last extraction, his bed sheet and pillow were "completely blood stained."

Fig. 1. Severe enlargement of the gingiva in the patient in case report with acute monocytic leukemia. The enlargement is due to the packing of leukemic cells in the submucosa. Petechia can be seen in soft palate.

For the past year, patient has had occasional epistaxis which were of short duration and never

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caused much blood loss. For the past few months, epistaxis have been somewhat more frequent and severe. The last nose bleed occurred on July 10, 1957 and, as a result of it, he sought a medical check up. He was told by his family physician then that he could find nothing wrong with him. His condition worsened and he went for medical attention to the New York Veterans Administration Regional Office. White blood cell count done at Regional Office on July 18, 1957 was 1,000 cells with a differential of two polymorphonuclear leucocytes, 88 lymphocytes, 10 monocytes. Patient's hemoglobin was 8.4 gm. with a red blood count of 2,600,000. Patient was referred for hospitalization with a severe anemia and a severe granulocyteopenia.

Past History: History was negative for dyes, employment hazard drugs, etc. and for carcinogenic producing agents (benzenes, etc.). He never used any paint sprays but loved to occasionally paint his own house. History of gonorrhea in 1908 and of rheumatic fever in 1910. Starting in 1934, patient had peptic ulcer symptoms and in 1947, a subtotal gastroectomy was successfully performed.

Family History: Patient came from Ireland and his entire family lived all their lives in Ireland. One brother died of cancer of the stomach, and several siblings died of unknown causes. There was no family history of tuberculosis, diabetes, hypertension, blood dyscrasias or anemia.

Physical Examination at First Admission: (July 19, 1957). Patient is a well developed, gray-haired white male who did not seem to be in acute distress nor chronically ill with a blood pressure of 130/65 and the pulse 78. His oral mucosa and conjunctiva were very pale, and there was moderate enlargement of gingiva which bled on touch. There were several small petechial hemorrhages on the left lateral aspect of the tongue. No lymphadenopathy was noted but ankle edema was present bilaterally. All other physical findings were essentially negative.

Laboratory and X-ray Data for July 19, 1957 Admission: X-ray examination was negative and other diagnostic tests were negative except for hematology tests.

During this first admission the patient's hemoglobin varied between 8.4 gm. and 9.1 gm. with a hematocrit of about 30%. White cell count varied from 1,400 to 4,800 and differential was usually 4 polys, 3 juveniles, 80 lymphocytes, 13 monocytes. Repeated urinalyses were negative. Bleeding time determinations were done several times and varied from 4 minutes to 8 minutes (normal bleeding time: 1-5 minutes). Coagulation time determinations were also done several times and were about 10 minutes (normal). Clot retraction time complete in 2 hours (normal). Platelet determinations varied from low of 90,000 to a high of 310,000 (300,000 normal). Prothrombin time was 15 seconds repeated several times with control of 13 seconds (normal). Bone marrow showed 40% of primitive lymphocytes. Other laboratory tests were essentially negative.

Course In Hospital: Patient was afebrile throughout his first hospital stay. His weight remained the same and hemoglobin dropped only slightly. He was transfused on August 30, 1957 prior to discharge with two units of whole blood. Patient's treatment in the hospital consisted of high caloric diet, multivitamins, and blood transfusions.

First Admission Final Diagnosis: The final diagnosis on his first admission was aleukemic phase of chronic lymphatic leukemia with agranulocytosis. He was discharged from the hospital on August 22, 1957, to be followed on a C.B.O.C. basis.

Physical Examination: Second V.A.H. Admission: (November 11, 1957) Patient seemed in moderately acute distress with dyspnea on minimal exertion and extreme tenderness of neck with blood pressure of 140/80 and pulse of 72. Conjunctiva and skin were very pale and he appeared chronically ill. Patient has several areas of ecchymosis on thigh, abdomen, left side of chest. These were multiple, bilateral, small enlarged nodes (neck region) which were very tender and easily palpable. These nodes were firm, freely movable, and walnut sized. Liver and spleen were both palpable and markedly enlarged. Slight flush of cheeks was seen bilaterally.

Multiple petechial hemorrhages were seen on palate, tongue, buccal mucosa and gingiva under denture. Patient's mucosa was very pale with tongue in midline. Gingiva around remaining lower teeth was heaped up and purplish in color. The gingival tissue was freely movable and painful to touch, and clotted blood was seen in the gingival sulcus.

Laboratory Data: Second Admission: White blood count, 112,000 with 49% lymphocytes, 14% monocytes and 51% lymphoblasts, platelets 40,000. Red

Fig. 2. Petechia on lateral borders of tongue of patient in case report.

blood count of 2,690,000 with a hemoglobin of 8.1 gm.

Total white blood count of 285,000 cells on November 25, 1957 (last count done).

Hospital Course: His hospital course this time was a rapidly deteriorating one and he expired on November 26, 1957. He was given supportive therapy, and put on 6-Mercaptopurine for last four days of his hospital stay.

Second Admission Diagnosis: Acute phase of chronic lymphatic leukemia.

LEUKEMIA

SUMMARY OF DISEASE

Leukemia is a disease of the white blood cells with an abnormal hyperplasia of the immature leukocytes. Thus, the white blood cells are like cancer cells in that they are unspecialized in function, do not mature and displace normal cells. This is a disease with a fatal termination and unknown etiology, characterized by a widespread systemic proliferation of the immature leukocytes and their precursors. The leukemic lesions are more or less widespread, involving primarily the blood forming organs, and then extending throughout the body to all organs and tissues. The disease occurs at all ages and in all races.

Etiology: The literature gives many causes that can result in the intracellular white blood cell deficiency that may produce leukemia. Excess radiation is listed as a chief cause of leukemia, and radiologists have ten times more leukemia than other physicians, being exposed to X-rays daily. There is also strong evidence that chronic benzene poisoning is an etiologic agent. The disease is considered by some investigators to depend on hereditary factors. Other evidence points to leukemia as a neoplastic disease. There are also references to viruses as a cause of leukemia, as it can be transmitted in birds and chickens from one to another.

Classification: Acute and chronic forms are distinguished by the number of blast forms (immature forms) present in the bone marrow, i.e., less than 10% is considered chronic case, 10-30% subacute case, over 30% acute leukemia case. In acute leukemia, the percentages of each maturation phase is very abnormal with the shift toward the extreme left. In chronic leukemia, the percentages of each maturation phase is normal. Subacute cases usually resemble acute leukemia clinically more so than the chronic variety.

Both acute and chronic cases of leukemia are then still further subdivided according to the predominating type of leukocyte or precursor found in the peripheral smear or bone marrow. One type of white cell involved are the lymphocytes and its precursor is the lymphoblast. Therefore, lymphatic leukemia is labeled lymphoblastic leukemia, and it refers to the cell of origin. Another type is monocytic leukemia, and it is referred to as myelogenous (Naegli) or histiocytic (Schilling) since the site or origin of monocyte is disputed. Still another type is myelocytic leukemia, or granulocytic, in which any one of the segmented (neutrophilic, eosinophilic, or basophilic) leukocytes are involved and is distinguishable according to predominating type of cell. In this type of leukemia, there may be

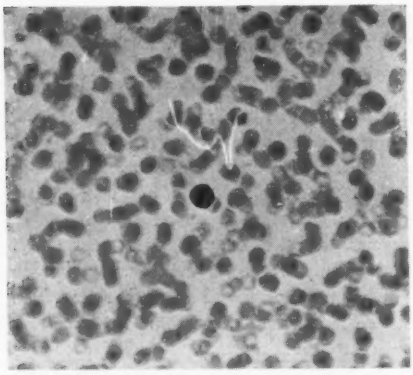

Fig. 3. 400X magnification of peripheral blood smear of Dr. A. M. Quart showing a mature, segmented, polymorphonuclear leukocyte surrounded by normal forms and numbers of red blood cells.

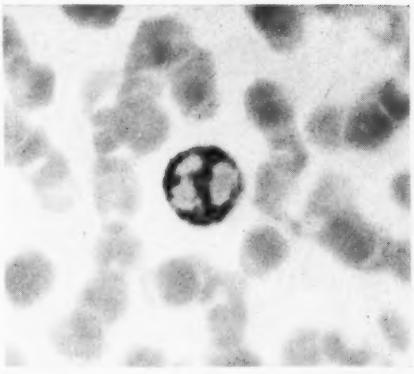

Fig. 4. 970X magnification of Fig. 3 illustrating the lobulations of a mature poly and normal number and morphology of red blood cell population.

a large number of mature forms of leukocytes in the peripheral blood in addition to myelocytes or myeloblasts. The most common variety is where neutrophilic myelocytes are the most numerous. There are great difficulties in differentiation of the various types of leukemia, especially in the acute stage, due to the extreme immaturity of the cells.

Aleukemic leukemia is a common term referring to cases of leukemia in which there is not only no leukocytosis (increase in peripheral white cells) but instead a granulocytopenia (reduction in peripheral white cells). The term which may be used with more understanding is sub-leukemic leukemia, which would mean a repression in numbers of the circulating leukemic cells (or immature cells) in the peripheral blood stream without an increase in total white cell count or there may even be a decrease in total white cell count.

Clinical Course: The onset of chronic leukemia may be so insidious that early diagnosis is usually difficult and most times discovered only on a routine blood examination. Early blood findings may be difficult to evaluate. Loss of weight, weakness, and anemia are the most common history findings. When the disease is sufficiently advanced to cause patient to seek attention, any of the following symptoms may be seen: anemia of various degrees with its various effects; enlarged spleen or liver or enlarged lymph nodes and their effects; loss of weight; bleeding, especially epistaxis or following tooth extraction; fever; and nervousness. Thus it can easily be seen that any of the body systems (gastro-intestinal, nervous, etc.) could be investigated before the true disease entity was diagnosed.

The onset of acute leukemia is sudden and the course is usually short. Acute leukemia is the commonest form of the disease in children and is more common up to fourth decade. Initial symptoms vary considerably for they depend on their site and development of the hemorrhage, leukemic infiltration, and glandular enlargements, as well as systemic manifestations such as fever, anemia, and toxemia. Fever, marked prostration, hemorrhagic manifestations, pallor, petechia or ecchymosis of skin, necrotic processes in the mucous membranes of the mouth and throat are usually the first signs. There is not as much adenopathy in the acute as in the chronic variant.

In lymphocytic leukemia adenopathy appears early and may be extensive and striking. The glands are discrete, moderately firm, freely movable and not attached to

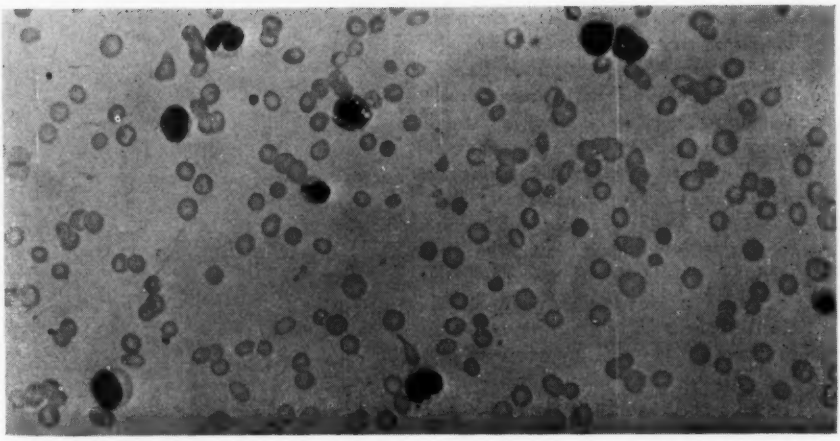

Fig. 5. 400X magnification of peripheral blood smear of patient in case report. Evidence is seen of poikilocytosis and anisocytosis of red blood cells. By comparison with Fig. 3 there are many more white cells and fewer red blood cells.

the skin. The glands of the cervical chain are usually most prominent but the post-auricular, and submaxillary glands may also be involved. Marked tonsillar enlargement which is asymptomatic may also be an early finding. Rarely may the salivary glands be involved by swelling. In monocytic leukemia the gingiva are grossly infiltrated and sometimes enlarge up to the incisal edges of the teeth.

Pathology: The distribution of lesions of bones in leukemia consists mainly of areas of diminished bone density (radiolucency). The ascending ramus of mandible is a site occasionally involved, and the cranial bones are also a common site. Radiolucency is seen in the marrow bones where the leukemic cells are usually the most active.

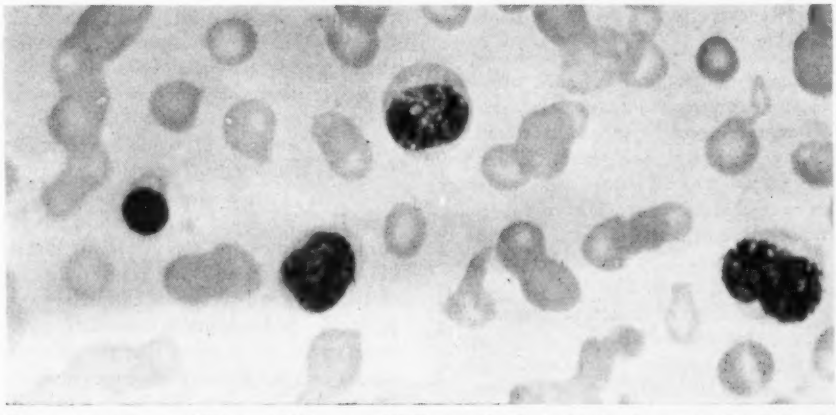

Fig. 6. Illustration of Fig. 5 at high power (970X magnification) showing in greater detail the red blood abnormalities. Lack of lobulations of the nuclei in the white cells represent immature cell forms.

Anemia, as stated before, is a common finding, and there are also morphologic changes in the red blood corpuscles. Poikilocytosis (variation in shape) is seen as well as anisocytosis (variation in size) in the red blood cell population. Increase in white cell count is the most important and significant diagnostic point with counts reported as high as 800,000 cells plus per cubic mm. A count of 100,000 plus is frequently seen, when the disease is just discovered. It is this increased production of white blood cells in the bone marrow which replaces the red blood corpuscle forming centers and results in anemia. Thrombocytopenia is also a characteristic finding, and frequently their number is below 100,000 platelets per cubic mm. By their increased numbers, the leukemic cells replace the megakaryocytes and thus platelet production is lowered. It is this reduction in platelet count that causes the bleeding in the leukemias. The most striking change in the white blood cell population is in the increased percentages of very immature cells (blast forms) in the peripheral blood. Bone marrow is just filled with these immature white cell forms.

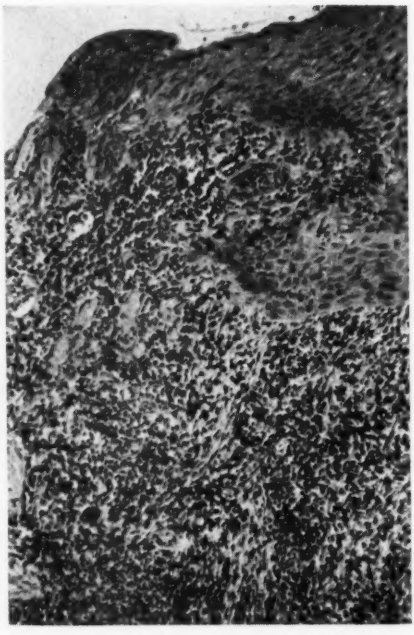

Fig. 7. 100X low power of gingival mucosa showing tremendous infiltration of immature leukemic cells in submucosa. Ulceration of mucous membrane is seen with no evidence of granulation tissue.

Infection and ulceration in various parts of the body are due to the decrease in the functioning white blood cells, especially the neutrophilic polymorphonuclear leucocytes. The immature white cells do not function as phagocytes or take part in antibody production.

Leukemia produces systemic effects by localized pressure in an evasive pattern to all organs, especially spleen, liver and other organs with lymphoid tissue. Thus there is infiltration of all tissues and replacement of normal architecture. Loss of function of an involved organ results and is due to the localized pressure of the leukemic cells in the involved organ. The degree of enlargement and loss of function depend some on cell type and duration but are due mostly to the great numbers of leukemic cell infiltration into the organ.

Oral Lesions: Petechial manifestations are characteristically found in the mucous membranes especially of the nose and oral cavity. "The first symptom may be unexpected bleeding following slight trauma, such as blowing the nose or brushing the teeth, and many patients find their way to the physician via the dentist or nose and throat specialist."[1] In addition to petechia, extensive ulcerations may be encountered. These are usually due to leukemic infiltrations and invasion of tissue by organisms found in oral cavity. Differential diagnosis here must be made with diphtheria, agranulocytosis, scorbutic gingivitis, Ludwig's angina and purpura, etc. "Partially clotted blood may be found at the gingival margins of the teeth and the gums themselves may be swollen and purplish in color. This swelling is sometimes so great that the gums are heaped over the teeth. There may be ulcerations and necrosis as well, the mucous membranes elsewhere in the oral cavity may be similarly involved, and there may be dif-

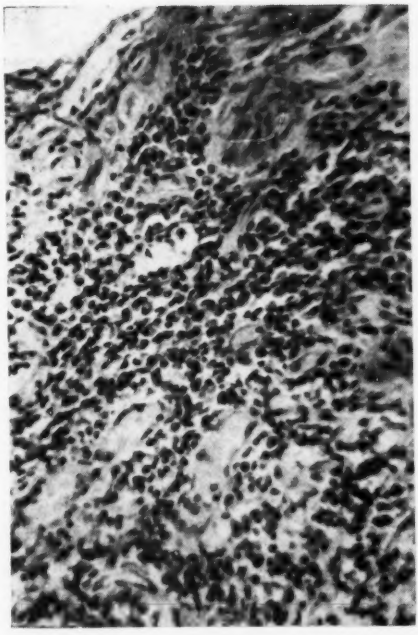

Fig. 8. High power of figure #7 showing areas of ulceration in greater detail. Primitive lymphocytes can be seen in the submucosa.

fuse cellulitis which extends into the deeper tissues of the face. It has been stated that swelling and ulceration of the mucous membranes, particularly the gingiva, is so characteristic of leukemia (mostly monocytic but also other variants) that this diagnosis should be suspected on clinical grounds."[2] The gingiva is characteristically swollen, of spongy consistency, and freely moveable. The description above applies in varying degrees to all acute leukemias.

Treatment: Treatment in leukemia is aimed at controlling or slowing mitotic rate of the immature white cells. Physical agents such as radiation (X-rays, radium or radioactive isotopes), and chemicals (radiomimetic) such as nitrogen mustards and myleran are used to control chronic leukemia. Cortisone and ACTH have a temporary beneficial effect especially in acute leukemic process, and the action here is a temporary reduction of immature cells.

Competitive chemical agents are used to control the numbers of immature cells in acute leukemia and the most common ones used are the folic acid antagonists, aminopterin and six mercaptopurine. Treatment of leukemia also consists of using as much supportive therapy as possible when symptomatically indicated and consists of: transfusions (whole blood), parenteral feedings, antibiotics, hormones, multivitamins, nutrients, analgesics, and sedatives, etc.

Histopathology of Lymphatic Leukemia In Gingiva: Thinning of epithelium may be seen with a dense infiltrate of leukemic cells in submucosa. The blood vessels seem to be surrounded by the leukemic elements. All of the stroma seems to be displaced by the infiltration and there may also be some hemorrhage seen in the submucosa. Ulceration is also seen but little or no granulation tissue is seen as the subepithelium is so crowded with leukemic cells.

The fundamental change pathologically is due to an extensive proliferation of the leukemic cells in the various tissues of the body and the blood stream.

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Papillon-Lefevre Syndrome: Precocious Periodontosis with Associated Epidermal Lesions

by JOHN I. INGLE, D.D.S., M.S.D.,* *Seattle, Washington*

THE recent paper by Dekker and Jansen, *Periodontosis in a Child With Hyperkeratosis Palmo - Plantaris*,[1] brings to light another interesting case of precocious resorption of the alveolar bone associated with epidermal lesions. This provocative syndrome, first described by Papillon and Lefevre in 1924,[2] has been reported primarily in Europe. A review of the literature reveals only three cases reported in North America.

In 1939 Corson of Philadelphia reported two cases of sibling children afflicted with "decided horizontal alveolar absorption".[3] He completed the description of their disease by stating, "Initially the gums began to show inflammation and the milk teeth soon became loose and fell out. With the second dentition there was again inflammation, and some of the second teeth loosened and fell out." Accompanying these severe oral symptoms was a punctate keratosis of the palmar and plantar surfaces with hyperhidrosis of the affected parts. Twelve years later, 1951, Rosenthal reported on the same two Philadelphia patients, tracing the oral advance of their disease as well as its epidermal course.[4]

In the interim, however, Woods and Wallace, in 1941, reported a similar case from Rochester, New York.[5] They adequately described the severe alveolar damage beginning with the primary dentition, but only casually mentioned the hyperkeratotic lesions of the hands and feet, and did not suggest the possibility of a definitive syndrome as did Papillon and Lefevre in 1924, or Corson in 1939. To my knowledge no new cases of the syndrome have been reported in the United States since 1941.

The recent article by Dekker and Jansen originated from Holland and dealt with a Dutch patient. Again the disease involved the primary dentition and was accompanied by severe acute inflammation. After reviewing the European and American literature Dekker and Jansen were able to report 19 patients with the syndrome. This presentation deals with the twentieth reported case of Papillon-Lefevre syndrome, hyperkeratosis palmo-plantaris with precocious periodontosis.

Fig. 1. Male patient, age 14. Right maxillary central incisor involved in periodontosis and drifted into severe labioversion.

CASE REPORT

On May 10, 1951, a 14 year old male of Scandinavian-Irish descent presented to the Periodontic Clinic at the University of Washington with the complaint, "a loose tooth". The maxillary right central incisor was truly loose—almost exfoliated, and had migrated to nearly horizontal position in labio-version. (Fig. 1).

Initial Examination

Further oral examination revealed a nor-

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Fig. 1a. Roentgenogram of right maxillary central incisor seen in Fig. 1, left. Pocket depth delineated by endodontic silver points placed to depth of pocket.

mal appearing gingiva except for a mild inflammation around the involved maxillary central incisor. (Fig. 2). Twenty-eight teeth were present with only a few small occlusal restorations and no active caries. On the other hand, examination with the periodontal probe revealed four deep infrabony pockets characteristic of periodontosis, mesial to the first permanent molars. (Fig. 3). A shallow pocket had developed between the mandibular central incisors. Radiographically, the thin, tapered root form of the mandibular incisors and first molars was apparent. (Fig. 4). An excellent Class I occlusion was present.

Apart from the oral lesions, the only abnormal findings were these:

a. Hyperkeratosis of the palmar and plantar surfaces with a tendency to fissuring and cracking of these zones. (Fig. 5).

b. A peculiar, dry, "dirty" skin on the dorsal surface of the arms, and the ventral surface of the legs. This skin, salmon-yellow in color, resembled "goose-flesh" or a nutmeg grater in appearance. (Fig. 6).

c. Generalized hypohidrosis.

d. Very fine body hair, greatly limited in amount for age 14.

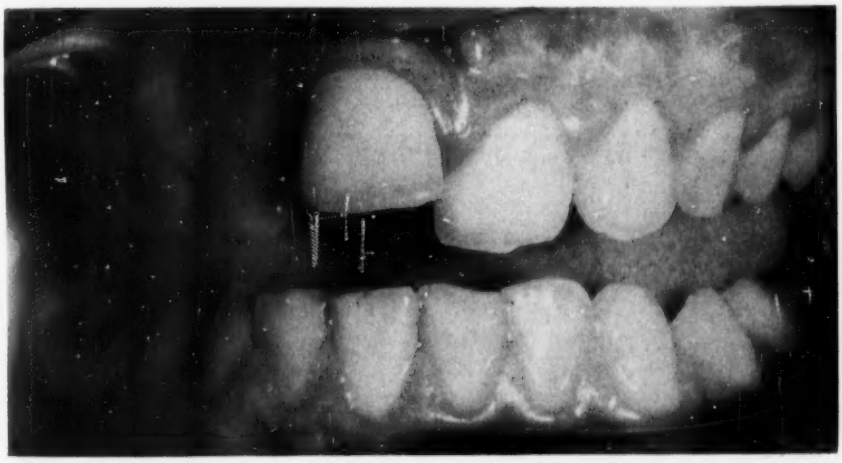

Fig. 2. Note the absence of gingival inflammation even apparent around the involved tooth.

Fig. 3. Clinical view of the left mandibular first molar region showing the absence of gingival inflammation associated with the involved tooth.

Fig. 3a. Roentgenogram showing initial infrabony periodontal lesion associated with the mesial of the left mandibular first molar. Note also the thin roots of the first and second molars.

Past History

The boy's mother reported he had had pertussis at age 4, chicken pox at age 6, mumps at age 6, and measles at age 10. At age 2 and again at age 4 or 5 she reported that his shoulder, elbow and wrist joints would "slip out". He had also experienced muscular or joint pain at age 7 or 8. His primary teeth had erupted and had been shed uneventfully. No gingival lesions had been noted.

The mother also reported that the involved incisor began to migrate when the patient was about 11 years of age. She further stated that her son had had a dry skin all his life, and at the age of 6 had experienced in the winter a period when his skin had chafed and cracked badly. The ends of his fingers had also cracked open at age 9 or 10.

The patient claimed to be athletic and further stated that after gym he did not have to take a shower like the other boys because he did not perspire.

There were two younger siblings, a brother and sister, and neither of these children had any skin or periodontal disorder. The mother and father did not have epidermal or periodontal disorders, nor did they know of any relatives or antecedents so involved. The parents were not consanguineously mated.

Physical Examination

A thorough physical examination by a pediatrician revealed no disease or abnor-

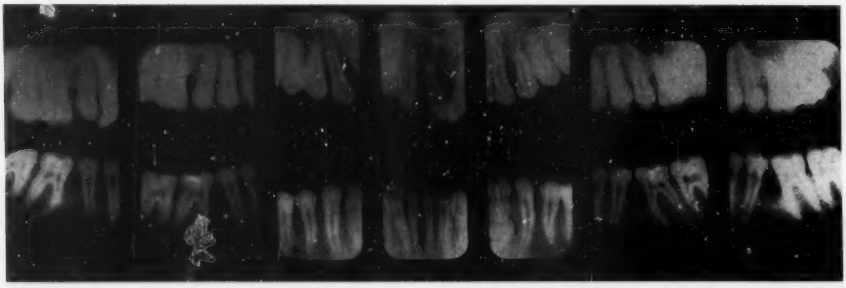

Fig. 4. Full mouth roentgenograms of a 14 year old male with periodontosis. Note the initial lesions associated with the maxillary central incisor and the mesial of all four first molars. The beginning lesion in the alveolar crest between the mandibular central incisors is also apparent, as is the thin root configuration of the first molars and mandibular central incisors.

Fig. 5. Hyperkeratosis of the palmar surface of a 14 year old male. The palm is extremely dry and resembles the hand of an old man.

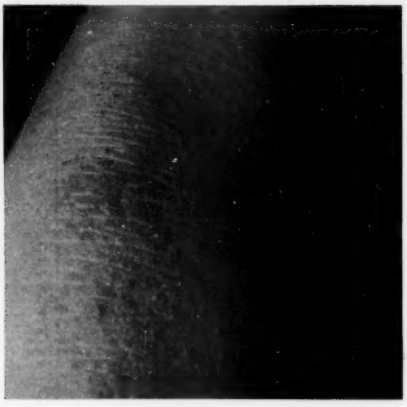

Fig. 6. Icthyotic appearance to the skin of the dorsal surface of the upper arm of a 14 year old male with Papillon-Lefevre syndrome. The scaly, piling up of hyperkeratotic epidermis is very apparent.

malities other than those noted at initial examination. Sexual development was well advanced. Although no facial hair was noted, fine axillary and pubic hair was in evidence. All joints functioned normally with no abnormal displacements. The heart sounds were entirely within normal limits.

Laboratory and Roentgenographic Examination

Hematology Report:

RBC—5.2 million

Hct.—40.7%

Sed. Rate—17.5 mm./hr. Wintrobe

Hb.—15.5 gms.

Erythrocytes — normochromic, anisocytosis 1 plus, poikilocytosis 1 plus

Platelets—normal

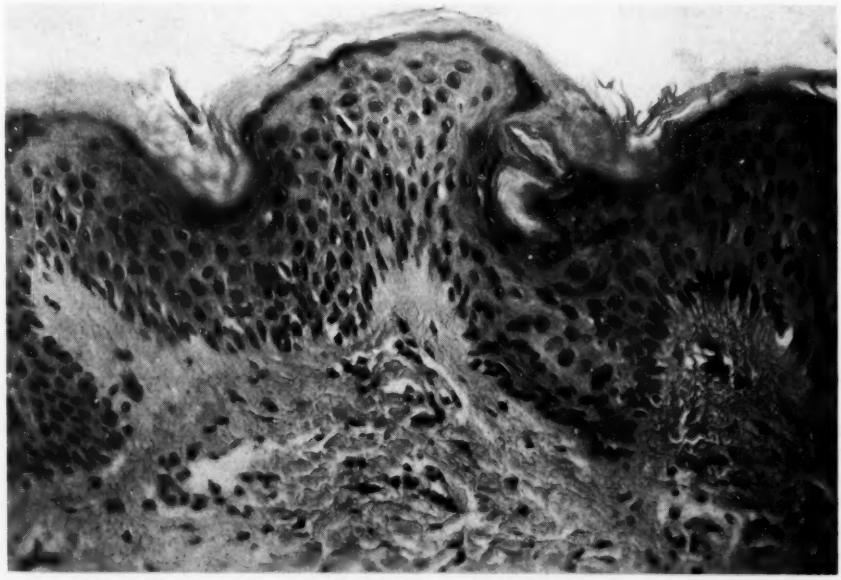

Fig. 7. This figure illustrates the upper half of the intra-epidermal segment of a sweat duct and attending limited poral keratosis. It will be observed that the granular layer is not detectable. This finding is compatible with the changes seen in icthyosis.

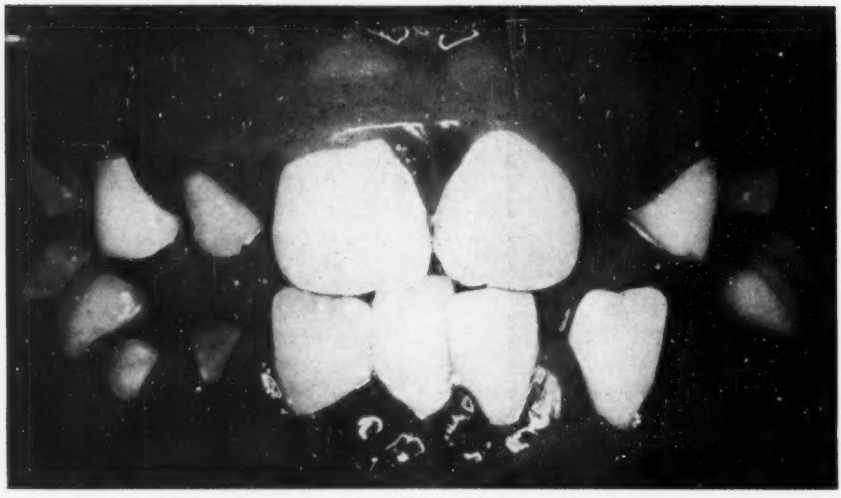

Fig. 8. Nine year old female suffering from childhood "periodontosis". The overwhelming gingival inflammation associated with this case immediately sets it apart from the previously illustrated male patient. Exfoliation of the primary teeth has developed before normal shedding occurred. (Photo courtesy Saul Schluger).

WBC—6,780
Differential count—normal

Chemistry
Serum—not fasting, lipemic
Calcium—10.6 mg.%
Phosphorus—4.1 mg.%
Alkaline Phosphatase—18.1 units.
(Found to be normal subsequently.)
Total protein—7.46 gms.%
Albumin—4.51 mgs.%

Urinalysis—essentially normal

Roentgenography—long bones and skull, normal

Dermatologic Examination

Initially a clinical dermatologic diagnosis of *pityriasis rubra pilaris* was made. Subsequent microscopic examination of a biopsy taken from the dorsal surface of the upper arm (Fig. 6) modified this diagnosis. The laboratory report* stated: "Sections of skin show minimal abnormality. Epidermal changes are characterized by a tendency to follicular plugging, adherence of closely packed lamellae of cornified cells and a discontinuous, frequently undetectable granular layer. These observations are compatible with, but hardly diagnostic of, *icthyosis*. Sweat ducts and sweat glands contained eosinophilic casts but were not cytologically remarkable." (Fig. 7).

*The author is indebted to Dr. George F. Odland for his interpretation of the microscopic sections of epidermis.

DISCUSSION

The childhood syndrome of alveolar atrophy with hyperkeratosis of the palmar and plantar surfaces is common to the case here reported as well as to the earlier cases of Papillon and Lefevre[2], Dekker and Jansen[1], Corson and Rosenthal[3,4], and Woods and Wallace[5]. It will be recalled that the cases reported by these earlier authors were accompanied by a violent gingival inflammation and severe destructive alveolar resorption beginning with the deciduous dentition. These prior cases more closely resembled the extreme alveolar atrophy in young patients unaccompanied by epidermal lesions as reported by Schluger[6] and Law[7]. (Fig. 8). In marked contrast to the previous reports, the case here reported was a classic periodontosis involving the permanent dentition. Only the

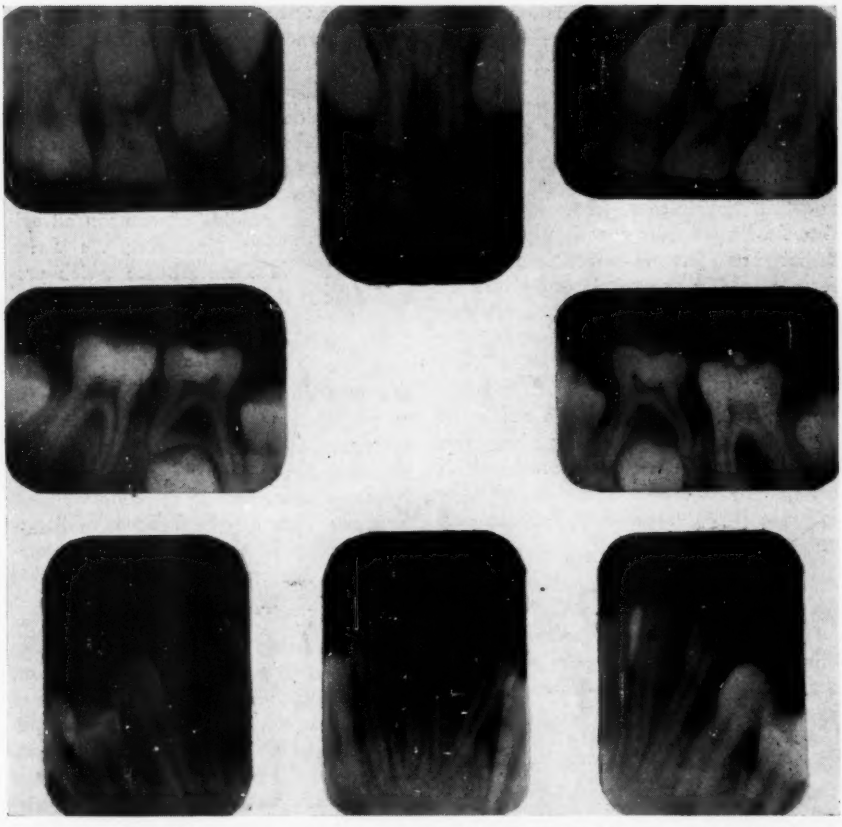

Fig. 8a. Full mouth roentgenograms of a nine year old female with childhood "periodontosis". The severe generalized alveolar resorption around the permanent incisors and primary teeth contrasts with the normal level of the alveolar crest around the permanent first molars.

alveolar process around the permanent first molars and incisors was involved, but these areas with no apparent inflammation. (Fig. 3 and 4).

In his recent text on Periodontics, Orban deals at some length with three possible types of periodontosis; (1) generalized periodontosis, (2) periodontosis involving a single tooth, and (3) periodontosis of the incisors and first molars[8].

The case here reported resembles more closely the "third group of periodontosis cases" outlined by Orban—young patients with vertical infrabony alveolar absorption related to the incisors and the mesial of the first permanent molars, tooth migration and diastemata, no caries, no calculus and no gingival inflammation. The previously noted cases more nearly resemble the "group one" cases of generalized periodontosis.

In seeking to explain the etiology of periodontosis, Orban has suggested a disturbance in the "functional interdependence between the tooth and the surrounding bone". He points out, as did McCall[9], the high incidence of enamel hypoplasia of the incisors and molars in the mouths of youngsters suffering from periodontosis. To McCall this suggested a possible dietary deficiency, especially vitamin D. To Orban, the enamel hypoplasia suggested a "pri-

mary disturbance in the function of the dental epithelium".

In an earlier article, Orban[10] has made a good case for the epithelial resting cells of Malassez as the stimulating mechanism in the formation of the cementoblasts and, thus, cementum. He points out that the so-called "resting cells" must have a function, for "it is hardly conceivable that epithelial tissue, having no function to perform, would remain vital throughout the life span of an individual". He further states, "These epithelial cells must undergo degeneration as they reach the end of their life span (20 to 30 days or slightly longer). To be found at all ages, these cells must regenerate by mitotic activity. However, such regeneration can be expected only if required by function." This function, he suggests, is the stimulation of continuous cementum deposition.

Orban also notes that, in addition to the frequency of enamel hypoplasia, Group III periodontosis cases often demonstrate unusually thin molar and incisor roots. This characteristic may also be related to a basic weakness in the original epithelial structures which made up Hertwig's epithelial root sheath, the "matrix" for root formation.

Orban summarized his hypothesis by stating[10], "The continuous formation of cementum is a necessary factor in functional adaptation. The pathogenesis of periodontosis (therefore) might have its basis in a developmental disturbance" of the epithelial structures.*

The Orban hypothesis of epithelial dysplasia as a factor in periodontosis fits very well the case here presented, as well as the cases described by Papillon-Lefevre and those following[11]. The fact that all cases of periodontosis need not be related to epithelial dysplasia becomes obvious but substantiates Orban's further contention that the different types of periodontosis have different etiologies. On the other hand, one might well argue that Group I, or generalized periodontosis, could as well be related to epithelial dysplasia as to a severe endocrine disturbance such as uncontrolled diabetes. One would also be justified in speculating on the possible effect upon all epithelial structures (including the dental epithelium) of a disturbance which produced gross skin lesions.

If a disturbance of the enamel epithelium was present in all these cases, why was enamel hypoplasia not apparent in all? In answer to this question, Orban has again speculated that situations may exist where the disturbance of the enamel epithelium is not extensive enough to produce enamel hypoplasia, but may be sufficient to disturb the function of the epithelial rests as the "trigger" for continuous cementum formation.

The greatest differences between the case here reported and the previously reported cases lie in the severe gingival inflammation, calculus formation and early loss of primary dentition suffered by the earlier cases but not in this present case. Moreover, this patient experienced hypohidrosis, whereas the other patients complained of severe sweating of the palms and soles. Furthermore, this is the only case definitely reported with skin lesions present other than the hyperkeratosis palmo-plantaris.

CONCLUSIONS

One may conclude that hyperkeratosis palmo-plantaris P-L (Papillon-Lefevre) is a recognizable syndrome involving skin lesions as well as oral lesions. Dentists should be urged to examine the palmar and plantar surfaces of patients suffering from periodontosis, and, by the same token, physicians should develop the habit of examining the oral cavity of patients presenting with hyperkeratotic palms and soles. The familial tendency and genetic pattern of this syndrome deserves further study.

*Zander and associates have reported the indirect relationship between apical cemental thickness and periodontal disease. Teeth severely involved periodontally exhibited thinner apical cementum than did teeth not periodontally involved.

SUMMARY

1. The syndrome hyperkeratosis palmoplantaris P-L (Papillon-Lefevre) is discussed.

2. A new case of hyperkeratosis palmoplantaris P-L with periodontosis is presented.

3. The possibility of epithelial dysplasia in the pathogenesis of periodontosis is explored.

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INTERNATIONAL ACADEMY OF ORAL PATHOLOGY

The International Academy of Oral Pathology will hold its first scientific conference in New York City on Monday, September 14, 1959 at the New York Academy of Medicine.

The morning session will be devoted to an analysis of the "Conditions that Predispose the Oral Mucosa to Cancer." Colonel Joseph L. Bernier, of the Armed Forces Institute of Pathology will serve as moderator, with Professor Martin Rushton, of Guy's Hospital, London, Professor Robert Bradlaw of the University of Durham, Newcastle upon Tyne; Doctor Lester R. Cahn of Columbia University, New York City, and Doctor Reidar F. Sognnaes, of Harvard University, Boston, acting as discussion leaders.

In the afternoon the subject of the conference will be the "Problem of Disturbed Odontogenesis in relation to Tumor Formation." Doctor Lester R. Cahn will serve as moderator with Colonel Joseph L. Bernier, Doctor Jens Jorgen Pindborg of the Royal Dental College, Copenhagen, Denmark, and Doctor Kurt H. Thoma, Professor Emeritus, Harvard University, acting as topic discussors. Doctor Harold Hillenbrand, Executive Secretary of the American Dental Association, and Doctor Henry A. Swanson, of Washington, D. C., are serving as consulting associates.

Attendance at the conference will be by invitation only.

Salivary pH Examination Amongst Indian Children and Its Relation to the Incidence of Periodontal Disease

by SANJANA, M. K., MEHTA, F. S., DOCTOR, R. H., SHROFF, B. C., *Bombay, India*

REPORTS vary much as to the reaction of saliva. Some state it is generally alkaline, others that it is practically neutral. Investigators like Starr[1] and others have found resting saliva to be slightly acid with the pH value ranging from 5.75 to 7.05.

Lothrop and Gies,[2] Marshall,[3] and Hopewell Smith[4] were among the first to try to find co-relationship between dental caries and the pH of saliva. While many investigators[5, 6, 7, 8, 9, 10] have tried to find whether a relationship exists between caries incidence and salivary pH value, references in the literature about investigations into the relationship of salivary pH and periodontal disease are almost nil. Hawkins[11] has tried to find out if any relationship existed between the periodontal disease and salivary pH.

Salivary pH studies in India have been very few, one of the most recent being by Shourie & Soni[12] and this also was carried out on adults. It was therefore deemed desirable to carry out a survey amongst school children, and also to determine whether any relationship is discernible between the pH value and incidence of periodontal disease amongst the individuals examined. Further it was desired to find out whether pH values varied with age within the age group examined. In India, the incidence of calculus deposit and periodontal disease have been shown to be high[13, 14] and the incidence of caries comparatively lower.[15] Shourie and Soni[12] found the average pH of stimulated saliva taken after lunch to be 7.38 in Indian adults.

This study was sponsored by the Gibbs Dental Research Scholarship, Bombay, India. Paper read at the 10th Annual Conference of the All India Dental Association.

*On the current exchange 150 Rs. equals about 30 dollars.

METHOD

735 Indian School Children between 11 to 16 years were examined. They came from the lower income group (earning up to Rs. 150/- per month) as well as the upper income group (earning Rs. 150/- and over). The saliva sample was stimulated saliva and collected by the open method, between 45 minutes and 90 minutes after meals and immediately examined, by using the glass electrode and Lumitron Photovolt pH meter.

Starr[1] suggested that the saliva should be collected under oil as the pH would change after collection. However, Brawley[9] examined pH of 3404 cases using colorimetric methods and found that readings taken immediately after collections did not vary so appreciably as to warrant collection under oil. At least three readings were taken on the pH meter of each sample and the mean of these three was recorded. The saliva collected was "stimulated saliva" the children being asked to chew paraffin wax to obtain sufficient quantities of the sample.

The oral examination was carried out in natural lighting with mouth mirror and probe. The data for periodontal disease was recorded on a card. The mouth was arbitrarily divided into six segments—each jaw consisting of a middle anterior segment from one canine to the other and inclusive of both canines and a right and left posterior segment. All clinical signs (swelling, redness, bleeding, pockets, etc.), were used to determine whether a papilla or margin was diseased or not. No X-rays were taken. Periodontal disease involving more than two papillae and margins in a segment was classified as being generalized for that segment. Otherwise the segment was considered to be locally affected. All cases were initially examined before inclu-

TABLE No. I

Age	*1 Mean pH for Upper Income Groups*	*2 Mean pH for Lower Income Groups*	*Combined Mean Average of 1 and 2*
11	7.62	7.37	7.49
12	7.57	7.35	7.46
13	7.49	7.38	7.44
14	7.59	7.44	7.51
15	7.62	7.33	7.47
16	7.66	7.39	7.52
Arithmetical Mean.	7.59	7.38	7.48

Average (M) Statistically found . . . 7.53.
Standard Deviation Statistically found . . . 3.298.
Standard Error of the Mean Statistically found . . . 0.122.

sion in the survey to exclude cases of traumatic bite, so that this factor would play a negligible role in the incidence of periodontal disease. Only cases generally affected by periodontal disease were considered as being affected for this investigation. Wherever used in this paper the term periodontal disease will include all departures of the gingival tissues from normal, except those deviations caused by specific infections.

The data collected is presented in the following tables.

Table I shows the mean pH for each age group from 11 to 16 years and for the lower income group children and the upper income group children separately. The grand mean for each class is also shown.

Table II gives the incidence of generalized periodontal disease amongst the various pH groups and the total number of children examined in each pH group.

In Table-III the incidence of generalized periodontal disease is given separately for the various segments examined. This table also shows the percentage incidence for each segment. *Analysis of the data:* It can be seen from the table that the grand mean pH for the lower income group is 7.38, whereas that for the upper income group is 7.59. The average pH for the lower income group is found to be consistently lower than the average pH for the upper income group of all ages. Also that there is a difference of 0.21 between the grand mean for the two groups ($7.59 - 7.38 = 0.21$). To test whether the difference is significant or not the students' test for small samples was carried out since we have only six

TABLE No. II

pH	*Total No. Examined*	*No. Not affected by Disease*	*No. affected by Disease*	*Percentage affected by Gen. Periodontal disease*
6.60 to 7.00	55	8	47	85.45
7.10 to 7.50	362	51	311	85.91
7.60 to 8.00	285	38	247	86.66
8.10 to 8.50	28	8	20	71.42

TABLE No. III

Percentage incidence of Generalized Periodontal Disease compared for various segments of the Mouth in 735 Children between the ages of 11 and 15.

	Anterior		*Left Posterior*		*Right Posterior*	
	Upper	*Lower*	*Upper*	*Lower*	*Upper*	*Lower*
Generally Diseased	499	516	501	401	456	357
Non-Generally Diseased	236	219	234	334	279	378
Percentage Diseased	67.89	70.20	68.16	54.56	62.04	48.57
Percentage not Diseased	32.11	29.80	31.84	45.44	37.96	51.43

age groups, viz., 11 years to 16 years. The value of it is seen to be 2.3701 which is not significant at 1% level but is significant at 5% level. So this is a borderline case and no definite conclusion can be drawn. One may conclude that although no conclusive proof is provided by the present evidence, there is a suggestion that pH value tends to be lower amongst the lower income group than for the upper income group.

Since statistically strong conclusive proof is not obtained that the difference between the lower income group and the upper income group is significant, the two groups are combined and the average mean pH calculated. The sample of 735 school children has been scientifically selected as far as possible on a random basis. By carrying out the statistical test the average (M) is found to be 7.53. This figure is not the arithmetical mean of the grand means for the upper and lower income groups. However, this statistically calculated average mean gives a truer picture as it allows for the inequality of the samples to be combined.

The standard deviation (σ) works out at 3.298 and the standard error of the mean (σ m) is calculated to be 0.122.

From here onwards the income group factor may be disregarded and the total number of children examined considered as a whole—since the income group factor has not been found to be definitely significant.

The coefficient of correlation (r) between age and pH was next worked out and found to be 0.08878 which means that there is no correlation between age and pH. Therefore, it may be concluded that practically the pH does not vary with age but is fairly constant among the age groups examined.

To find whether there is any relationship between pH and generalized periodontal disease the Chi-square test (x^2 test) is applied. The value of x^2 works out 4.2387. For $n = 3$ this value is not significant. One concludes that there is no relationship between pH and generalized periodontal disease.

The constituents of the diets consumed by the individuals examined were almost similar except that meat was added in small amount in a few individuals. Hence no separate recording was made of the diets in relation to pH.

The T test of difference between percentages for the incidence of generalized periodontal disease is next carried out (a) comparing the upper anterior segment versus the lower anterior segment and (b) comparing the upper right and the lower right posterior segments and (c) comparing the upper left and lower left posterior segments. For the anterior segments the value of T is 0.9581, which is not signif-

icant. For the left posterior segments the value of T works to be 5.4 which shows significance. Similarly for the right posterior segments the value of T is 5.1947 which again is significant. We conclude that while there is no significant difference between the upper anterior and the lower anterior segments in the incidence of generalized periodontal disease, there is a significant differene between the upper left posterior and the lower left posterior segments. Similarly there is a significant difference between generalized periodontal disease incidence for the upper right segment compared with that for lower right segment. The lower posterior segments are more healthy.

SUMMARY

1. The salivary pH was investigated amongst 735 Indian School Children between the ages of 11 years and 16 years.

2. The saliva sample was stimulated saliva and collected by the open method, between 45 minutes and 90 minutes after meals and immediately examined by the glass electrode method.

3. While the findings suggest a pH value variation for lower and upper income groups children, no conclusive proof was obtained statistically.

4. The statistical average mean salivary pH was found to be $7.53 \pm (2.58 \times 0.122)$.

5. There is no correlation between age and pH value.

6. There is no relationship between pH of saliva and generalized periodontal disease incidence.

7. The incidence of generalized periodontal disease is significantly greater for the upper posterior segments of the mouth than for the corresponding lower posterior segments.

8. No significant difference was found for the generalized periodontal disease incidence for upper and lower anterior segments.

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Epidemiology of Diabetes Mellitus: I. Review of the Dental Literature*

by R. C. SHERIDAN, JR., D.D.S.;** E. CHERASKIN, M.D., D.M.D.;*** F. H. FLYNN, B.S., M.T., A.S.C.P.,**** AND A. C. HUTTO, B.S., M.T., A.S.C.P.,****
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THE literature is replete with articles showing that diabetes mellitus is a common disorder. A number of diabetes detection surveys have been made in the general population as well as in selected groups such as the military service, the aged, the obese, and among industrial employees. Interestingly enough, only *one* survey has been recorded in a dental population—in other words, among individuals who present themselves in a dental clinic because of some oral problem or simply for routine or periodic dental examination. This is worthy of note because, according to leading medical authorities,[1] the oral manifestations of diabetes mellitus rank among the most important findings.

On the other side of the ledger, one can note that approximately fifty articles have appeared in the dental literature during the past twenty years concerned directly with the oral manifestations of diabetes mellitus. Very little of the latter information of the past two decades has been in quantitative form and there has been little integration of the observations of the different investigators.

The purpose of these reports, therefore, will be to attempt to answer the following *four* questions:

1. What is the prevalence of diabetes mellitus in a dental population? In other words, how often does an *unrecognized* diabetic patient appear in the dental office?

2. What is the prevalence of oral findings of diabetes mellitus in a dental population? In other words, what symptoms and signs will the *unrecognized* diabetic patient appear with when he presents himself for oral examination and care?

3. Among nondiabetic patients, is there any correlation between blood glucose levels and oral symptoms and signs? In other words, can one observe relationships in the oral cavity to justify the term *prediabetes mellitus?*

4. What is the prevalence of the antithesis of diabetes mellitus? In other words, what are the oral findings in patients with *flat* glucose tolerance patterns?

This report will simply review the available literature pertaining to the questions which have been raised. A report to follow[2] will attempt to answer the above questions from a study of 100 routine dental patients.

REVIEW OF LITERATURE

The literature pertinent to this study may be divided into *two* categories: (1) the criteria for the detection of diabetes mellitus, and (2) the oral manifestations of diabetes mellitus.

Criteria for the Detection of Diabetes Mellitus: A number of epidemiologic studies have been done in the general population as well as in selected groups. These will be considered separately.

General Population Studies: Various methods have been used for screening purposes as well as for the definitive establishment of the diagnosis of diabetes mellitus. Generally speaking, most of the epidemiologic studies performed among the general

*This investigation was supported in part by a traineeship grant (2G-15) from the Epidemiology and Biometry Section, Public Health Service.

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population have utilized either: (1) *urinalysis*, (2) *blood sugar* or *glucose*, or (3) a *combination of urinalysis and blood sugar or glucose* as the screening procedure.

Urinalysis as the Screening Procedure: Three studies of considerable size have been reported in which urinalysis was done as the screening test.

Sharkey et al[3] studied 69,159 individuals (Table 1). The Galatest was utilized. Where positive tests were obtained by the powder method, the patients were also checked by the Clinitest and Benedict methods. An incidence of 4.7 percent of the patients showed melituria. These were recalled for blood sugar determinations. Only 2,024 of the 2,991 with positive urine samples received subsequent blood testing. Of this number, 151 were known diabetic patients and 148 cases of previously unrecognized diabetes mellitus were found. In other words, 0.20 percent of the individuals tested during this diabetic detection drive in Dayton, Ohio, were found to suffer with previously unrecognized diabetes mellitus. The mass testing of Dayton school children during this survey revealed a significant number of newly discovered cases of juvenile diabetes. Of the 38,528 children tested, melituria was noted in 4.8 percent of the cases. Among the 1,022 of the 1,867 school children with positive urine samples who had subsequent blood sugar determinations 18 (1.7 percent) previously unrecognized cases of diabetes mellitus were discovered. Thus, .05 percent of the children tested were found to have previously unknown diabetes mellitus.

TABLE I

Percentage of new diabetic patients discovered in the general population with the use of urinalysis as the screening method

author(s)	*no. of patients screened*	*percentage of new diabetics*
Sharkey et al[3] (1950)	69,159	0.20
Albrink et al[4] (1951)	1,025	0.19
Loube and Alpert[5] (1954)	2,657	0.30

Albrink, Parente and Gelperin[4] analyzed (Table 1) the results of a diabetic survey in Greater New Haven, Connecticut (a population of 241,542). The total number of persons responding to the drive was 1,025 (.5 percent of the population). Thirty-two of those tested (3.1 percent) were reported to have melituria. All persons showing sugar in the urine were referred to their private physicians for further study. The final diagnosis of the thirty-two patients disclosed eight previously known diabetic patients, seventeen false positive reports, five lost in the follow-up, and two (.19 percent) new diabetic patients.

During the Diabetic Detection Week in 1951, a total of 19,828 urine specimens were tested, of which 866 (4.4 percent) were reported positive. Loube and Alpert[5] studied a segment (2,657) of this large group (Table 1). The sample was utilized because of the consistency in obtaining and evaluating the urine specimens. All of the analyses had been checked by means of the Galatest powder procedure. Of the 2,657 urine specimens included in this group, 220 (8.2 percent) were found to be positive for sugar. To each of the 220 individuals with a positive test, a letter was sent so that the patient could be further investigated. Of the 220 persons who were sent questionnaires, 174 (79 percent) responded. Some of the patients visited their private physicians and others reported to the laboratory of the investigators for glucose tolerance tests. The conclusion was drawn that approximately 0.30 percent were new diabetic individuals.

Table 1 summarizes the findings from these detection surveys based upon urinalysis for the detection of glucose in the blood. It can be seen that the figures for the different studies are essentially the same. One can also note that the percentage of unrecognized diabetes mellitus according to this detection method is small.

Blood Glucose as the Screening Procedure: Petrie, McLoughlin, and Hodgins[6]

studied 241,457 persons (Table 2). Each of these individuals received a glucose determination on the venous blood by the Anthrone method.[7] If the blood glucose proved to be 160 mg. percent or over, the patient was referred for a subsequent glucose tolerance test. If the individual had not eaten previously, a blood glucose of 120-159 mg. percent was regarded as adequate justification for a glucose tolerance examination. The glucose tolerance test consisted of a fasting blood glucose and determinations at 45 minutes and two hours after the ingestion of 100 grams of glucose. Of these 241,457 persons receiving complete tests, 1.87 percent were referred to their private physicians because of above normal or borderline carbohydrate metabolism.

Urinalysis plus Blood Sugar as the Screening Procedure: Wilkerson and Krall[8] examined 3,516 of the 4,983 inhabitants of Oxford, Massachusetts. The urine was tested for sugar by the standard Benedict's qualitative test. If positive, the specimen was examined by a quantitative Benedict's test. In adult patients the venous blood sugar was measured according to the Folin and Wu method[9]. In the case of children, the capillary blood specimen was checked for sugar by the Folin-Malmros[10] microsugar technique. The screening level for hyperglycemia was selected as 160 mg. percent for venous blood and 190 mg. percent for capillary blood. If a patient showed any melituria or if the blood sugar proved to be above the levels just described, both the urine and blood were re-checked. If the results of the second test were above the diagnostic level, a diagnosis of diabetes mellitus was made. If a discrepancy showed between the initial and the second test, a dextrose tolerance test was performed. A total of 70 cases of diabetes mellitus was found. This included 40 previously known cases and 30 newly discovered diabetic patients. Thus, 0.8 percent new diabetic patients were recognized during this survey (Table 3).

Harwood[11] tested the urine for sugar by means of the Galatest in men and the Clinitest in women in 1,957 individuals. Capillary blood sugar was simultaneously determined by the Wilkerson-Heftmann method.[12] A significant hyperglycemia was regarded as 160 mg. percent or greater. He concluded that 5 percent of new diabetic patients were recognized during this survey (Table 3).

TABLE II

Percentage of new diabetic patients discovered in the general population with the use of blood sugar as the screening method

author(s)	*number of patients screened*	*percentage of new diabetics*
Petrie et al[6] (1954)	241,457	1.87

Studies in Specific Groups: A number of different studies are available based on surveys of *selected* groups of the population. In some instances the screening procedure was simply an urinalysis and in other instances both urine and blood sugar determinations were employed.

Urinalysis as the Screening Procedure: Gates[13] studied 1,800 individuals in an industrial plant who for years had had careful, thorough physical examinations including at least one urinalysis per year (Table 4). Seventy-one individuals gave a positive family history of diabetes mellitus and 46 showed melituria while 8 were known diabetic patients. Glucose tolerance tests were performed on all those with a positive hereditary background and those who displayed melituria. Nine of those

TABLE III

Percentage of new diabetic patients discovered in the general population with the use of urinalysis plus blood sugar as the screening method

author(s)	*number of patients screened*	*percentage of new diabetics*
Wilkerson and Krall[8] (1947)	3,516	0.8
Harwood[11] (1953)	1,957	5.0

TABLE IV

Percentage of new diabetic patients discovered in a selected group of the population with the use of urinalysis as the screening method

author(s)	*nature of population*	*number of patients screened*	*percentage of new diabetics*
Gates[13] (1942)	industrial employees	1,800	0.90
Blotner and Hyde[14] (1943)	selectees and volunteers	45,650	0.30
Spellberg and Leff[15] (1945)	selectees	32,033	0.02
MacBryde[17] (1949)	aged, obese, history of family diabetes	17,451	0.90
Rochon and Moran[18] (1957)	dental patients	3,122	1.20

with a family history and 11 with sugar in the urine proved to be diabetic. Thus, this investigator found an incidence of 0.9 new diabetic patients in this select population of 1,800 industrial employees.

Blotner and Hyde[14] studied 45,650 consecutive selectees and volunteers between the ages of 18 and 45 who appeared for final examinations before induction into the armed forces (Table 4). Urinalysis was routinely done, and the urine checked for sugar by Benedict's qualitative solution. If urinalysis proved positive, the classical three-hour glucose tolerance test was done and the venous blood sample measured according to the method of Folin and Wu.[9] These investigators found an incidence of 0.8 percent melituria. If any of the blood samples during the glucose tolerance test reached 180 mg. percent and some or all of the urinalysis showed sugar, then the individual was regarded as diabetic. According to these standards, 0.3 percent of the selectees and volunteers were found to be suffering from diabetes mellitus.

Spellberg and Leff[15] studied the urine of 32,033 consecutive selectees processed at the New Orleans induction station (Table 4). All individuals with positive urine samples were subjected to an Exton-Rose[16] glucose tolerance test. In this manner, 9 cases (0.02 percent) of diabetes mellitus were noted.

MacBryde[17] checked the urine of 17,451 individuals, principally in the old age category, obese persons, and those of diabetic families, in the city of St. Louis, Missouri (Table 4). Melituria was observed in 3.46 percent of the cases by means of the Galatest method. Where possible, blood was drawn two hours after the last meal, and true blood glucose was determined. Whenever the blood glucose sample proved to be 120 mg. percent or more true glucose, the patient was considered to be diabetic. This investigator concluded that there was an incidence of 0.9 percent new diabetic patients.

Rochon and Moran[18] routinely examined the urine by the Clinitest procedure of every new dental patient at the University of Detroit School of Dentistry during a 5-year period (Table 4). The 3,122 ambulatory dental patients ranged in age from 12 to 80. Forty-three (1.5 percent) diabetic patients were detected during this study. Only 5 of these 43 persons knew that they had been suffering with diabetes mellitus. Consequently, these investigators found an incidence of 1.2 percent previously unrecognized diabetic patients. The followup on these urinalyses was done by referring the patients to their private physicians.

Urinalysis plus Blood Sugar as the Screening Procedure: Canelo, Bissell, Abrams, and Breslow[19] studied 945 employees at 4 industrial plants (Table 5). Urinalysis was performed by means of the Benedict test. The blood sugar values were determined by the standard Folin-Wu[9] method. Patients

who showed melituria or a blood sugar of 160 mg. percent or over (130 mg. percent if the subject had eaten more than 2 hours previously) were informed that they should consult their private physicians for further examination. The screening urinalysis revealed 5.0 percent patients with positive sugar in the urine and 8.4 percent with a trace of melituria. Precisely what was done by the local physicians to establish a diagnosis of diabetes mellitus is not clear. However, these investigators reported the detection of six previously known and nine not previously recognized cases of diabetes mellitus, giving a total of 0.9 percent new cases.

Kenny, Chute, and Best[21] examined 4,419 persons in all age groups except the preschool category (Table 5). Urine samples were tested qualitatively for reducing substance by means of the Clinitest method. True blood glucose was measured in capillary blood samples according to the Somogyi-Nelson method.[22,23,24] All measurements were made immediately after breakfast or lunch. If the blood glucose values were in excess of 160 mg. percent between 1 and 1½ hours after a meal and the values proved to be 150 mg. percent between 1½ and 2 hours or the levels were over 120 mg. percent 2 hours after a meal or in a fasting state, the screening tests were considered abnormal. Such persons and those with melituria were subjected to a 2-hour glucose tolerance test. A diagnosis of diabetes mellitus was made if the blood glucose values proved to be 200 mg. percent or more on two occasions or during the glucose tolerance test. Diabetes was also regarded as being present if both the peak was above 200 mg. percent and the 2-hour test greater than 120 mg. percent. A fasting blood sugar of more than 120 mg. percent was also considered as evidence of diabetes mellitus. These investigators claimed a total incidence of 0.5 percent previously unrecognized diabetic patients.

TABLE V

Percentage of new diabetic patients discovered in a selected group of the population with the use of urinalysis plus blood sugar as the screening method

author(s)	*nature of population*	*number of patients screened*	*percentage of new diabetics*
Canelo et al[19] (1949)	industrial employees	945	0.9
Tabor and Frankhauser[20] (1950)	over age 40	550	2.9
Kenny et al[21] (1951)	above preschool age	4,419	0.5
Harting and Glenn[25] (1951)	above age 25	3,186	2.2
Kenny and Chute[27] (1953)	above the age of 6	6,673	0.5
Carroll et al[28] (1954)	above age 16	5,708	0.4
Chesrow and Bleyer[29] (1955)	nondiabetic above age 60	1,000	6.4

Tabor and Frankhauser[20] studied the blood sugar determinations by the Folin-Malmros procedure[10] of 550 patients over the age of 40 (Table 5). Those individuals with blood sugar levels above the screening level (150 mg. percent) were requested to return for followup studies. Fasting blood sugar determinations and urinalysis were done. Those with a fasting sugar level of greater than 120 mg. percent and those with a fasting melituria were asked to have a sugar tolerance test. A total of twenty-two cases of diabetes mellitus was discovered. This included six previously known and sixteen newly discovered cases. This number represents an incidence of four percent among this group with a percentage of 2.9 newly recognized cases.

Harting and Glenn[25] studied 3,186 persons over the age of 25 by means of urinalysis (Galatest procedure) and capillary

blood sugar (Folin and Malmros method).[10] Most of the subjects presented themselves for the initial test with preparatory instructions. Preliminary screening levels were selected from the initial blood sugar readings. Patients were recalled for further determinations if the initial blood sugar was 160, 150, 140, or 130 mg. percent ½ to 1½, 1½ to 2½, 2½ to 3½ or more than 3½ hours later respectively. All patients with melituria were also recalled. Of the 3,186 patients, 10.6 percent showed a blood sugar greater than the established screening levels, and 14.1 percent of the subjects had melituria. These individuals were recalled, at which time capillary and venous blood specimens were taken and each analyzed by the Folin-Malmros,[10] Folin-Wu,[9] and Somogyi-Nelson[26] methods. Of the 3,186 persons over 25 years of age examined, 71 (2.2 percent) newly discovered diabetic patients were detected by using both blood sugar and urine sugar as the original screening procedure.

Kenny and Chute[27] studied the prevalence of diabetes mellitus in two Ontario towns (Table 5). A total of 6,673 persons over the age of 6 were tested as reported earlier.[21] They found 0.5 percent new diabetic patients.

Carroll, Kurlander, and Nester[28] examined 5,708 individuals above the age of 16 by means of urinalysis (Clinitest) and blood sugar (Wilkerson-Heftmann tablet method[12]) using 180 mg. percent as the critical threshhold (Table 5). However, later in their study they used values above 130 mg. percent as indicative of diabetes mellitus. According to their calculations, there were 0.4 percent previously unrecognized patients with diabetes mellitus.

Chesrow and Bleyer[29] studied 1,000 patients ranging in age from 60 to 110 (Table 5). Between 2 and 2½ hours after a meal, venous blood was drawn and the sugar concentration measured by the Wilkerson-Heftmann tablet method.[12] The reducing substances in the urine were determined qualitatively with Clinitest tablets. Patients whose blood sugar screened above 130 mg. percent or who had a trace or more of reducing substances in the urine were given a glucose tolerance test of the 3-hour type. In all blood samples of the glucose tolerance test, the true blood sugar was determined by the Somogyi[23] and Folin-Wu[9] procedures. These investigators reported an incidence of 6.4 percent previously unrecognized diabetic individuals.

ORAL MANIFESTATIONS OF DIABETES MELLITUS

A study of the dental literature for the past twenty years discloses a list of about fifty publications directly related to diabetes mellitus and the oral cavity. Those of particular importance in this discussion may be divided into *three* groups: (1) *oral symptoms*, (2) *oral signs*, and (3) *oral roentgenographic evidence* of diabetes mellitus.

Oral Symptoms: The most common subjective complaints reported in the literature are: (1) *dry mouth*, (2) *burning mouth*, (3) *tender gingiva*, (4) *pain with percusson of the teeth*, and (5) *dry sockets*.

Dry Mouth: Frequent reference is made to the association of xerostomia and diabetes mellitus. Sheppard[30] examined a group of 100 diabetic patients including 12 under the age of 21 and 4 above 70 years of age. This author divided the diabetic individuals into 3 categories which he referred to as mild, moderate, and severe diabetes. However, no mention is made in his report as to the criteria for this classification. Sheppard reported dryness of the mouth as constant or almost so in 6 percent of the cases, occasionally present in 20 percent, and only rarely observed in 8 percent. Banks[31] and Amies and Park,[32] without citing figures, also reported the association of xerostomia and diabetes mellitus.

Burning Mouth: Sheppard,[30] in his study of 100 diabetic patients, made mention of the fact that 2 percent of his cases complained with a burning sensation in the oral cavity.

Tender Gingiva: Rudy and Cohen[33] studied 403 diabetic patients. They simply

mention the fact that gingival tenderness is a common complaint. Rutledge,[34] in his study of 20 diabetic children between the ages of 8 and 19 years, also made mention of the presence of tenderness of the gingiva. Lovestedt and Austin[35] studied the incidence of periodontoclasia in a group of 503 diabetic and 1,023 nondiabetic patients and observed the symptom of gingival tenderness. Amies and Park[32] analyzed the oral findings in 32 diabetic children age 6 to 14 years attending a diabetic camp and made casual mention of tenderness of the gingiva. Kaplan,[36] Rudy and Cohen,[37] and Aboul-Ela[38] made similar observations without citing specific data.

Pain with Tooth Percussion: Rudy and Cohen[33,37] Kaplan,[36] Rutledge,[34] Pollack, Person, and Knishkowy,[39] and Martinez[40] observed sensitivity of the teeth to percussion. However, the frequency of this symptom nor its method of appraisal are not considered in any of these reports.

Dry Sockets: Banks[31] mentioned the fact that alveolar osteitis occurs with greater frequency in diabetic patients. Krongold[41] indicated specifically that dry socket occurs more often even in patients with controlled diabetes mellitus. Zerbe[42] reported that alveolar osteitis is twice as frequent in diabetic as against nondiabetic patients.

Oral Signs: The most common oral areas discussed in connection with diabetes mellitus are: (1) *lips,* (2) *gingiva,* (3) *tongue,* and (4) *teeth.*

Lips: Martinez[40] mentioned, without citing figures or criteria, the frequent occurrence of dryness of the lips.

Gingiva: A number of interesting gingival signs of disease have been observed and described in diabetic patients. They include: (1) *swelling,* (2) *bleeding,* (3) *violaceous hue,* (4) *pocket formation,* and (5) *recession.*

Gingival Swelling: Rudy and Cohen,[33,37] Kaplan,[36] Rutledge,[34] Lovestedt and Austin,[35] Banks,[31] Pollack, Person, and Knishkowy,[39] Amies and Park,[32] and Aboul-Ela[38] all have mentioned the common occurrence of gingival edema. Ziskin, Siegel, and Loughlin[43] specifically indicated a 15 percent incidence of spongy gingiva in their series of cases.

Gingival Bleeding: Rudy and Cohen,[33,37] Kaplan,[36] Rutledge,[34] Lovestedt and Austin,[35] Banks,[31] Amies and Park,[32] Martinez,[40] and Aboul-Ela[38] have all indicated the common association of easy gingival bleeding and diabetes mellitus. Ziskin, Siegel, and Loughlin[43] specifically indicated a 32 percent occurrence in the 94 children in their series.

Violaceous Gingival Hue: Ziskin, Siegel and Loughlin[43] indicated gingival discoloration in 92 percent of their series of 94 diabetic children. Pollack, Person, and Knishkowy[39] and Aboul-Ela[38] simply commented on the presence of a violaceous hue to the gingival tissues without establishing the frequency.

Pocket Formation: Ziskin, Siegel and Loughlin[43] cited an incidence of 89 percent of diabetic children with periodontal pockets. Banks[31] simply mentioned the association.

Tongue: A number of interesting lingual signs of disease have been observed in diabetic individuals. They include: (1) *smoothness of the dorsum of the tongue,* (2) *geographic pattern,* (3) *tongue coating,* (4) *redness,* (5) *enlargement,* and (6) *fissures of the tongue.*

Smooth Tongue Surface: Sheppard[30] pointed out that migratory areas of denudation on the dorsal surface of the tongue may be associated with diabetes mellitus.

Coated Tongue: Sheppard[30] reported that the most common oral finding was that of coated tongue in 92 percent of his cases. However, he felt that this finding is insignificant in his chronically ill hospitalized series of patients.

Red Tongue: Sheppard[30] and Banks[31] simply pointed out the association between

diabetes mellitus and lingual erythema.

Enlarged Tongue: Sheppard[30] and Banks[31] claimed macroglossia as a diabetic finding.

Fissured Tongue: Sheppard,[30] Banks,[31] Pollack, Person, and Knishkowy[39] and Martinez[40] reported fissuring of the tongue as a sign of diabetes mellitus.

Teeth: A number of interesting dental observations have been made and reported in the literature. They include: (1) *increased tooth mobility*, (2) *increased tooth loss*, and (3) *caries experience*.

Increased Tooth Mobility: Rudy and Cohen,[33, 37] Kaplan,[36] Rutledge,[34] and Martinez[40] all reported loosening of the teeth.

Tooth Loss: Lovestedt and Austin[35] reported a greater incidence of tooth loss in diabetic patients than in their observed nondiabetic population.

Caries Experience: Whether there is increased dental caries or not in the diabetic individual is still an open question. Krongold,[41] and Cohen[44] claimed an increase in dental caries. In contrast, Ziskin, Siegel, and Loughlin,[43] Banks,[31] Sindoni,[45] and Aboul-Ela[38] concluded that there is no increase in the caries experience.

Roentgenographic Findings: The two most discussed roentgenographic findings are: (1) *alveolar bone atrophy*, and (2) *marginal widening of the periodontal membrane*.

Alveolar Bone Atrophy: Rudy and Cohen,[33] Kaplan,[36] Pollack, Person, and Knishkowy,[39] Stahl,[46] Golomb,[47] Martinez,[40] and Person[48] all have claimed that diabetes mellitus is associated with increased bone atrophy. Rudy and Cohen,[37] in a subsequent report, disagreed with their own earlier report and claimed no bone loss. Rutledge[34] observed bone loss in 10 of 20 children in his group. Lovestedt and Austin[35] claimed that there was no significant roentgenographic difference in a group of 503 diabetic and 1,023 nondiabetic patients.

Marginal Periodontal Widening: Rutledge[34] observed this finding in 14 of the 20 diabetic children in his study. Rudy and Cohen[37] also noted this change. However, Ziskin, Siegel, and Loughlin[43] could not demonstrate marginal periodontal widening in their series of cases.

DISCUSSION

Several points are worthy of mention in the review of the detection of diabetes mellitus. First, the evidence suggests that the more elaborate the screening procedure, the greater the detection of previously unrecognized diabetic patients. This is borne out by a comparison of Tables 1, 2, and 3. By simply utilizing the examination of urine samples, the percentage of new diabetic patients is about 2 per 1,000. By means of blood sugar screening procedures, the figure rises to almost 2 per 100. By the use of blood sugar and urine techniques, the incidence increases to 5 per 100. Secondly, it is evident that the more select the sampling of the population, the greater the differences in the incidence of unrecognized cases. Thus, the higher the age group, the greater the number of previously unrecognized cases (Table 5). For example, Kenny et al[21] and Kenny and Chute[27] found only 0.5 percent in groups excluding only preschool children. In contrast, Harting and Glenn,[25] Tabor and Frankhauser,[20] and Chesrow and Bleyer[29] found increasing numbers of diabetic patients as older groups were studied. Thirdly, the more rigid the requirements for diabetes mellitus, the smaller the percentage of positive screenees will prove to be true diabetic patients. Finally, mention should be made of the fact that but one study is reported in a dental population,[18] and it is based only on urinalysis as the screening technique.

It is interesting to observe that a number of oral findings have been associated with diabetes mellitus. These, as reported earlier in this paper, may be divided into: (1) oral symptoms, (2) oral signs, and (3) oral roentgenographic pathoses.

There is no question but that it is extremely difficult to quantitate symptoms since their presence is largely derived from the patient's statements. None the less, one cannot ignore the presence of symptoms, and it is of note that a number of investigators have attempted to relate various complaints to diabetes mellitus. Thus, from the review of the literature it appears that approximately 6 percent of diabetic patients complain with constant dry mouth and 20 percent with occasional xerostomia. Presumably, 2 percent of diabetic individuals report burning mouth. Apparently, dry sockets seem to occur twice as often in diabetic versus nondiabetic patients.

Signs of disease are somewhat easier to measure since the examiner has the opportunity of observing them directly. Only rarely have investigators attempted to quantitate changes in the oral cavity with the diabetic pattern. Gingival swelling has been reported in 15 percent of one series of cases. Gingival bleeding has been claimed to be present in approximately one-third of a group of diabetic children. One group of investigators have noted a violaceous hue of the gingiva in approximately 92 percent of their series of diabetic individuals.

The two most commonly discussed roentgenographic findings are alveolar bone loss and marginal periodontal widening. There is still no agreement as to whether these findings occur. In one study 50 percent of a group of children were observed to have abnormal alveolar bone loss. This same investigator reported 70 percent of a group of diabetic children with marginal periodontal widening.

It is clear from the review of the literature and this brief discussion that the evidence seems to indicate that oral findings are associated with diabetes mellitus. However, most of the publishable material must be recognized as being in the realm of opinion rather than fact. It would be extremely helpful to observe a group of patients who present themselves for routine dental care and to study the frequency with which the previously described oral symptoms and signs and roentgenographic findings occur. This will be described in the report to follow.[2]

SUMMARY

1. The evidence from leading medical authorities seems to indicate that some of the most commonly occurring symptoms and signs of diabetes mellitus appear in the oral cavity.

2. During the past two decades approximately 50 articles have appeared in the dental literature dealing with the oral manifestations of diabetes mellitus. Very little of the material is of a quantitative nature and, therefore, it is difficult to draw any definite conclusions.

3. Of the many epidemiologic studies performed in diabetes mellitus, only one has been reported in a dental population. This particular study utilized urinalysis as the screening procedure.

4. The evidence is quite clear that the more sophisticated the screening technique, the greater the detection of diabetes mellitus.

5. A report to follow[2] will analyze the oral clinical and roentgenographic findings in the light of the three-hour true glucose tolerance test.

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UNIVERSITY OF MINNESOTA

The University of Minnesota School of Dentistry is most pleased to announce the 3rd Annual Course in Advanced Oral Pathology for 1960. The course held from Thursday, 21 April through Tuesday, 26 April 1960, devotes about half time to examination of approximately 200 slides. Among the subjects and lecturers will be:

1) Salivary Gland Pathology—Dr. John T. Godwin, Professor of Pathology, Emory University, Atlanta, Georgia.

2) Bone Pathology—Dr. Louis Lichtenstein, Chief of Pathology, Veterans Administration Center, Los Angeles, California.

3) Advanced Roentgenographic Diagnosis of Diseases of the Jaws—Dr. Edward Stafne, Professor Emeritus, Mayo Clinic, Rochester, Minn.

4) Roentgenographic Technics for the Head and Neck—Dr. Richard W. Moss, Chief, Oral Surgery Section, Veterans Administration Center, Wood, Wisconsin.

5) Dermatopathology—Dr. Robert Goltz, Professor of Dermatology, School of Medicine, University of Minnesota, Minneapolis, Minnesota.

6) Odontogenic Tumors—Dr. Robert J. Gorlin, Professor of Oral Pathology, School of Dentistry, University of Minnesota, Minneapolis, Minnesota.

7) Oral Manifestations of Systemic Disease—Dr. Anand Chaudhry, Professor of Oral Pathology, School of Dentistry, University of Minnesota, Minneapolis, Minnesota.

The course will be limited to 20 participants. Tuition will be $100.

Interested individuals will please contact: Dr. Robert J. Gorlin, Professor and Chairman, Division of Oral Pathology, School of Dentistry, University of Minnesota, Minneapolis 14, Minnesota.

Influence of Injury and Adaptation of the Periodontal Ligament to Pathologic Changes During Experimental Lathyrism

by ALVIN F. GARDNER, D.D.S., M.S.,* PH.D.,** *Baltimore, Md.*

GROWING interest in the pathologic physiology of connective tissues and in the treatment of connective tissue disorders has in recent years directed the attention and energies of the medical world towards the control of the functions and reactivity of connective tissues.[1-11] This study is an attempt to gain an insight into the functions and reactivity of a specialized connective tissue known as the periodontal ligament.

In the present state of our knowledge, this connective tissue is termed the periodontal ligament. The reasons for this terminology are several fold. This connective tissue is anchored to the bony socket by numerous bundles of collagenous as well as by acid resistant fibers which react with aldehyde fuchsin after peracetic acid oxidation. It is, therefore, similar to ligaments in other areas of the body, since it contains stress bearing fiber bundles called the principal fibers, and also loose connective tissue between these fiber bundles containing blood vessels, lymph vessels and nerves.

To establish the casual relationship between lathyrism and the changes observed in the experimental animals, supplementation with dietary proteins and amino acids was instituted. It is the purpose of this investigation to determine the protective action of dietary proteins and amino acids against experimental lathyrism with particular emphasis placed upon the alterations in the periodontal ligament, a specialized connective tissue.

An insight into the diseases influencing the growth of the periodontal ligament presupposes an understanding of the metabolism of connective tissue.

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EXPERIMENTAL MATERIALS AND METHODS

Ninety-six male albino weanling rats, 45-50 gms. in weight, of the Holtzmann-Sprague-Dawley strain, were used throughout this investigation. The rats were divided into groups at weaning and placed on various diets containing sweet peas. An additional group of male rats were pair fed and served as normal littermate controls. Each of the experimental rats was paired with one of the normal controls of the same weight. The rats were housed in an open-bottom mesh cage. All control animals were sacrificed at the same time of day as the experimental animals. The rats were separately caged in an isolated room maintained at the usual laboratory temperature (70-80° F.). The animals were weighed periodically and their weight gains were recorded. Immediately after death the rats were autopsied. The carcasses were skinned and eviscerated. The organs were examined for gross changes. The skulls were removed, x-rayed and finally fixed in Zenker-formalin solution for one day, or in Bouin's solution for two days. These fixatives were employed as they preserve the desired histologic details excellently, resulting in good morphologic differentiation. Individual bones to be histologically examined were treated as described under the sections on histologic preparation and analysis.

GROUND LATHYRUS ODORATUS DIETS

The *Lathyrus odoratus* diets were prepared by grinding the whole dry peas to a fineness which permitted all the pea meal to pass through a 24-mesh sieve. The dry ingredients were mixed separately and stored, the cod liver oil being added to portions of the mixture as needed.

The feedings were continued for different periods utilizing the various experimental and control diets which are described in detail below. For diets 1 to 7, the feedings were continued for one, two, three, four and five week periods respectively. For diets P.D. I to P.D. VII, the feedings were continued for 56 days.

The experimental diet was as follows:

1. Lathyrus odoratus meal 50%, casein 10%, cornstarch 25%, sucrose 5%, brewer's yeast powder (dried) 4%, salt mixture (Hubbell, et al.[12] 1937) 4% and cod liver oil 2%.

The control diets were as follows:

1. This control diet contains the same ingredients as in diet 1 of the experimental diets but replacing the Lathyrus odoratus meal by 50% dried and ground commercial edible pea meal.

2. Milled Rockland rat diet.

The protective diets (Lathyrus odoratus 30%, plus dietary supplements) were as follows:

P.D.* I. This is a control diet for all protective groups, no supplements being added. It consists of ground sweet peas 30%, casein 10%, starch 45%, sucrose 5%, brewer's yeast 4%, salt mixture (Hubbell, et al. 1937) 4%, and cod liver oil 2%.

The following are experimental diets plus supplements:

P.D. II. Ground sweet peas 30%, casein 40%, starch 15%, sucrose 5%, brewer's yeast 4%, salt mixture (Hubbell, et al.) 4% and cod liver oil 2%.

P.D. III. Ground sweet peas 30%, gelatine 25%, sucrose 5%, starch 30%, brewer's yeast 4%, salt mixture (Hubbell, et al.) 4% and cod liver oil 2%.

P.D. IV. Ground sweet peas 30%, casein 35%, glutamine 5%, cysteine Hcl 4%, sucrose 5%, starch 11%, brewer's yeast 4%, salt mixture (Hubbell, et al.) 4% and cod liver oil 2%.

P.D. V. Ground sweet peas 30%, casein 10%, glutamine 5%, sucrose 5%, cysteine Hcl 4%, starch 36%, brewer's yeast 4%, salt mixture (Hubbell, et al.) 4% and cod liver oil 2%.

P.D. VI. Ground sweet peas 30%, casein 10%, sucrose 5%, cysteine Hcl 4%, starch 41%, brewer's yeast 4%, salt mixture (Hubbell, et al.) 4% and cod liver oil 2%.

P.D. VII. Ground sweet peas 30%, casein 10%, glutamine 5%, starch 40%, sucrose 5%, brewer's yeast 4%, salt mixture (Hubbell, et al.) 4% and cod liver oil 2%.

HISTOLOGIC PREPARATION AND ANALYSIS

After sacrifice and fixation the upper and lower jaws were dissected free from the skulls, so that tissue sections consisted of upper molars, upper incisors, lower incisors with lower molars. These were embedded separately in individual blocks.

Mesiodistal sections through two bucal roots of the upper and lower molars, buccolingual sections of the upper and lower molars, sagittal sections of the upper and lower incisors, and frontal sections of total intact hemisections of skulls were prepared for sectioning, mounting and staining by the various methods employed in this investigation.

All histologic sections were cut consecutively at 6 to 8 micra and stained with hematoxylin and eosin, Mallory's phosphotungstic acid-hematoxylin, van Gieson's stain, Periodic acid-Schiff stain, Wilder's stain or impregnated with silver according to the method of Gomori. After mounting in Canada balsam, the cover slips were weighted down to obtain flat sections.

*P.D. is used as an abbreviation for protective diets.

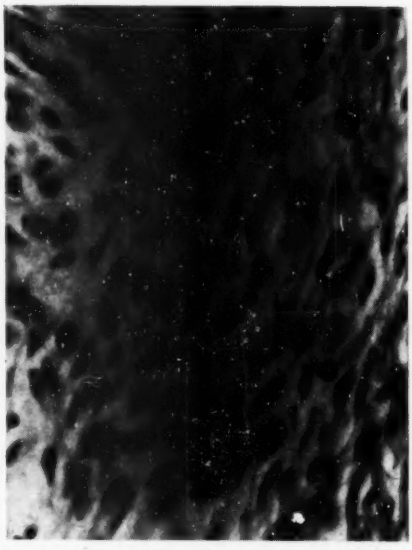

Fig. 1. Photomicrograph of the periodontal ligament from a male rat fed a Rockland rat diet (littermate control) for 8 weeks. Note that most of the cells of the ligament are typical young fibroblasts. They are long, stellate connective tissue cells whose nuclei are large and oval in shape. They lie at the surface of the collagen fiber bundles and are, probably, active in the formation and maintenance of the principal collageneous fibers. (Hematoxylin and eosin stain; orig. mag. X750.)

FINDINGS

During the course of investigation of changes in mesodermal tissues during experimental lathyrism, it became readily apparent that a major site of alteration existed in the periodontal ligament.

Experimental Molar Periodontal Ligament After 8 Weeks of Protective Diets. In protective diet I the main feature which gave the periodontal ligament a peculiarly ominous appearance was the structure of the fibroblasts as they lay in rows between large areas of homogeneous, eosinophilic, hyaline appearing tissue which they were forming. In juxtaposition to this homogeneous eosinophilic material the fibroblasts became enlarged and hyperchromatic as they assumed some of the characteristics of osteoblasts. The presence of a homogeneous eosinophilic zone with palisading of enlarged fibroblasts within the periodontal ligament was specific only for ex-

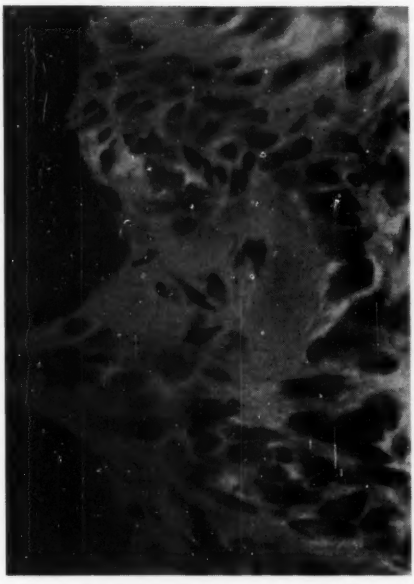

Fig. 2. Photomicrograph of the periodontal ligament from a male rat fed a 30 per cent sweet pea diet for 8 weeks. Note the initial location of the collagen deposits and rarity of fibroblasts in the hyaline areas. Nests or rows of cells are embedded between layers of hyalin. (Hematoxylin and eosin stain; orig. mag. X800.)

perimental lathyrism and will be referred to as "lathyritic bodies" (Fig. 2). In all animals fed protective diet I an overwhelming number of lathyritic bodies were located throughout all areas of the periodontal ligament (Figs. 9 and 10). They were just as numerous at the apex as at the alveolar crest or at the bifurcation of molar roots. The lathyritic bodies occupied primarily the middle one-half of the width of the periodontal ligament being separated from the cementum and alveolar bone by a thin zone of connective tissue.

There was a decided difference in the morphology of the fibroblasts of the periodontal ligament in animals fed nonsupplemented sweet peas as compared to normal control animals (Fig. 4). Comparison of the fibroblasts in the periodontal ligament of normal and sweet pea fed animals revealed those in the sweet pea group (P.D. I) to be larger, plump, with more abundant granular cytoplasm and large, ovoid, vesicular nuclei with nucleoli, while those in the normal controls were smaller, with less cytoplasm and smaller spindle-shaped nuclei with a dense chromatin network. The nuclei of the fibroblasts from protective group I animals were hyperchromatic and eccentrically located in the cell.

This diffuse spindle cell growth of fibroblasts with palisading may be regarded as a basket cell proliferation of fibroblasts, or as an aggregate of narrow cylindrical palisade cells.

The thickness of the periodontal ligament in all animals of this group was less than in the controls and the number of fibroblasts per unit areas was likewise decreased. Along with this decrease in the number of fibroblasts per unit area and the difference is their appearance, there finally was an increased number of collagen fibers and intercellular material per unit area in the ligaments of protective group I animals. The blood supply of animals fed protective diet I was relatively scant.

Epithelial remnants so common in control periodontal ligaments were absent in protective diet I animals. Acute and chronic inflammatory cells were absent in all areas of the periodontal ligament. The physiologic distal drift with differentiation of peripheral periodontal cells into osteoblasts and osteoclasts was disturbed and distal migration of the teeth had ceased.

The majority of the principal fibers of the periodontal ligament which normally run in a more or less continuous fashion from bone to cementum, were disrupted as they approached and entered the lathyritic bodies. These fibers were clear and distinct on either the bony or cemental side of the lathyritic bodies but became masked immediately upon entering the homogeneous eosinophilic areas. Cells of the periodontal ligament were frequently found trapped within the homogeneous masses (Fig. 3).

In protective diet group II the lathyritic bodies which were peculiar to experimental lathyrism were still present, however, their number and distribution was altered (Fig. 11). There was a significant reduction in the total number of these lathyritic bodies in animals of this group and they appeared more numerous in the apical one-half of the ligament. The gingival one-half of the ligament contained scattered lathyritic bodies but no continuous distribution which was so evident in all animals fed protective diet I. However, the morphology of the lathyritic bodies was similar to that found in protective group I, except that palisading of cells were not as prominent. Vascularity was reduced but only in the apical one-half of the ligament was there a significant reduction in the number of capillaries.

Epithelial remnants were reduced in the apical region, where the lathyritic bodies occupied significant portions of the ligament. The width of the periodontal ligament was slightly decreased. The distal drift of the molars was present with osteoblasts and osteoclasts differentiating properly along the periphery of the ligament with bone formation and resorption in more or less balance. In the animals given protective diet III, very minimal numbers of lathyritic bodies were present. A few isolated bodies occurred in the most apical portion of the ligaments. Palisading of cells were absent in the periodontal ligaments of this group. Ankylosis was consistently missing in all animals of this group. Vascularity was normal in most areas of the ligament, decreasing wherever lathyritic bodies occurred.

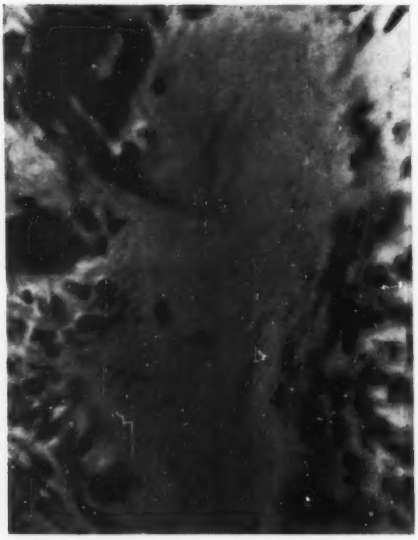

Fig. 3. Photomicrograph of the periodontal ligament from a male rat fed a 30 per cent sweet pea diet for 8 weeks. Note the large dense collageneous deposits with a few cellular inclusions typical of the lesions of lathyrism. The majority of cells bordering the collagen deposit are proliferating fibroblasts. These deposits show all the staining reactions of collagen, and an internal fibrillar structure is visible at this magnification. (Hematoxylin and eosin stain; orig. mag. X750.)

Epithelial remnants were similar in quantity and distribution to normal control animals. The width of the periodontal ligament was within the normal range and the physiologic distal drift of the molars was progressing satisfactorily.

In the animals fed protective diet IV, lathyritic bodies were reduced in number, and those remaining were principally located in the most apical regions.

In the animals fed protective diet V, the lathyritic bodies were reduced in quantity and palisading of cells was minimal compared to P.D. I group. These bodies were located only in the apical one-third of the ligament.

In the animals given protective diet VI, a decreased number of lathyritic bodies were present and palisading was decidely reduced in comparison to P.D. I group. The distribution was primarily confined to the apical one-half of the ligament.

In the animals fed protective diet VII, lathyritic bodies were reduced in number as well as in the degree of palisading of cells in comparison to protective diet I group. The bodies that persisted were primarily in the apical one-half of the ligament.

Experimental Molar Periodontal Ligament After 1, 2 and 3 Weeks of 50 Per Cent Sweet Pea Diets. In the sweet pea fed groups the main features were similar to those which produced the peculiar ominous appearance so specific for experimental lathyrism. However, the degree of alteration mainifested was dependent upon the duration of time that the animals received the lathyrus pea. Thus the influence of the lathyrus toxin on the periodontal ligament appears to be cumulative in nature.

In the group of animals fed a 50 per cent sweet pea diet for one week there was an absence of the pathognomonic lathyritic bodies so evident in protective diet I animals. In the periodontal ligament of these weanling rats no difference in the number and morphology of the fibroblasts could be observed (Fig. 11). If there was any significant difference at this stage in the experimental animals, the fibroblasts were slightly decreased per unit area and were more spindle-shaped. The number of collagen fibers per unit area was slightly greater in the experimental animals of this group than in the littermate controls. In addition, occasional small foci of an eosinophilic material containing fine disoriented fibrils were only occasionally seen in the experimental animals principally in the apical one-third. The fibroblasts in the periodontal ligament of this group were well preserved and in most cases were diffusely dispersed in the stroma of the ligament.

In the group of animals given a 50 per cent sweet pea diet for two weeks growth was poor. The fibroblasts of the periodontal ligament produced a lattice work of spindle-shaped cells interspersed with collagen fibers, forming a loose connective tissue in palisade arrangement. These dense layers of spindle-shaped cells lay on the outer surfaces of an eosinophilic material which occurs in the form of a lake or body. The periodontal fibroblast bordering these hyaline lakes were bizarre, large cells and the tissue had an overwhelmingly prominent pattern of a differentiated connective tissue in which collagenous fibers were readily produced. Many large, bizarre cells were found initially at the apex but after the second week of the experimental sweet pea diet the growth of pleomorphic cells became more extensive and advanced in an incisal direction.

The vascularity of the ligament in the majority of animals of this group was moderately reduced due to replacement of numerous areas of the ligament by an eosinophilic nonvascular material (Fig. 7). Epithelial remnants had apparently degenerated in the regions of the lathyritic bodies and were generally reduced throughout the ligament. The width of the ligament in all animals of this group was reduced in comparison to the littermate controls. The physiologic distal drift was absent with an increase in resorption and an altered osteoblastic-osteoclastic relationship (balance). The principal fibers of the periodontal ligament were disrupted in all areas where the presence of the lathyritic bodies occurred. The fibers were seen as definite and distinct entities (bundles) on either side of the lathyritic bodies but shortly upon entering the

eosinophilic material they became completely lost or masked in the homogeneous substance of this material.

In the periodontal ligament of animals fed sweet pea diet III for a period of three weeks, the growth was very poor. The histologic pattern of the normal-appearing littermate (Fig 1) had been completely replaced in all areas of the ligament by the lathy-lathyritic body. The lathyritic body consisted of densely cellular palisading tissue surrounding acellular, avascular, homogeneous eosinophilic bodies (Fig. 6). The palisading cells consisted of many large cylindrical hyperchromatic cells, with single, large nuclei containing nucleoli. Dividing cells were not common throughout the ligament, and mitotic figures were absent among the palisading cells. The transformation of normal fibroblasts into abnormal ones in the periodontal ligament occurred without the addition of any known carcinogen. It is not unexpected that periodontal ligament cells are capable of this kind of transformation when the lathyrus toxin combined with mechanical stress were operative. The lathyritic bodies were now equally distributed throughout all regions of the ligament so that the peculiar ominous appearance was similar whether one examined the bifurcation area, the apical area or the gingival region of the ligament.

The normal vascularity of the periodontal tissue was markedly reduced due to the degree of avascular tissue in all regions of the ligament. Epithelial remnants were absent from all regions. Evidently they had been replaced by proliferating fibroblasts and homogeneous eosinophilic material (Fig. 8). The width of the ligament was decidedly reduced throughout its entire supporting surface. The physiologic distal drift of the molars was arrested due to decreased osteoblastic activity and increased osteoclastic activity. The majority of the principal fibers in the periodontal ligament of the animals of this group showed an interruption in their continuity. This disruption appeared to occur primarily in the middle of the ligament. Dystrophic calcification was initially developing within the lathyritic bodies in a few animals of this group. The principal location for this occurrence was in the gingival one-third of the periodontal ligament not far from the alveolar crest. It appeared that a tendency existed for osseous metaplasia to occur in these lathyritic bodies via the intramembranous route. The calcified material developed within the central zone of the eosinophilic lathyritic body (Figures 5, 5A, and 5B).

Experimental Incisor Supporting Structures After 8 Weeks of Protective Diets. In protective diet I the main features which gave the periodontal ligament and labial alveolar periosteum a peculiar appearance were the presence of small eosinophilic lakes and cementoid material or dystrophic calcification respectively. In the alveolar fibers of the periodontal ligament small irregular homogeneous, eosinophilic lakes occurred in the basal or middle third, however, no typical lathyritic bodies were

Fig. 4. Photomicrograph of the periodontal ligament from a male rat fed a 30 per cent sweet pea diet for 8 weeks. Note the marked degree of hyalinizing fibrosis in this ligament during lathyrism. The hyaline material is strongly eosinophilic and separated by rows of palisaded fibroblasts. The nuclei of the palisaded fibroblast is eccentrically located and hyperchomatic. (Hematoxylin and eosin stain; orig. mag. X1200.)

evident. The lakes occurred between groups of fibroblasts, however, no palisade arrangement of cells occurred at the periphery of the lakes. The intermediate plexus showed edema and some disorientation of cells. The cemental zone was normal. The labial alveolar periosteum was free of inflammatory cells and was widest at the basal zone where a folding of the enamel had occurred. Toward the anterior one-half of the incisor, there was an edematous increase in intercellular substance. At the level of the alveolar crest cells were scarcely distributed throughout the ground substance.

The blood supply was very abundant with dilated and hyperemic blood vessels throughout the entire periosteum which were particularly concentrated adjacent to the folded enamel. The posterior portion of the labial alveolar periosteum had lost some of its fibrous appearance and assumed more of an edematous character. At the most basal end of the labial alveolar periosteum opposite the folding of the outer enamel surface, irregular small and large bodies were present resembling abortive cementoid formation. This cementoid material or dystrophic calcification appeared somewhat similar to the cementum which was produced on the surface of the dentin. However, there was an absence of cementoblasts in this area, therefore, the bodies are best referred to as cementoid

or dystrophic calcification since they probably arose from cells of the labial alveolar periosteum. The basal or fundic connective tissue contained dilated and hyperemic blood vessels.

In protective diets II, III, IV, V, VI, and VII the peculiar ominous appearance of eosinophilia among the alveolar fibers was no longer present.

Experimental Incisor Supporting Structures After 1, 2 and 3 Weeks of Sweet Pea Diets. In the animals fed a 50 per cent sweet pea diet for a period of one week, no significant changes were present in the periodontal ligament, labial alveolar periosteum or in the basal or fundic connective tissue.

In the animals given the same 50 per cent sweet pea diet for two weeks no discernible changes were evident beyond the range of normality in either of the three divisions of the incisor supporting structures, i.e., periodontal ligament, labial alveolar periosteum or fundic connective tissue. In sweet pea group III which was fed the 50 per cent diet for three weeks the main feature evident for the first time was an initial disorientation of cells in the alveolar zone. No palisading of cells occurred nor was eosinophilia present in the ground substance in any area of the periodontal ligament. The basal or fundic connective tissue contained many dilated and hyperemic blood vessels.

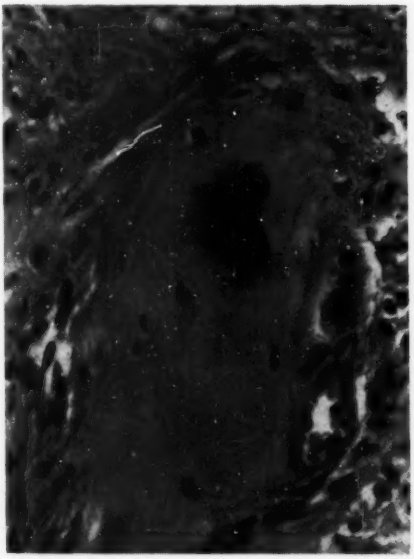

Fig. 5. Photomicrograph of the periodontal ligament from a male rate fed a 30 per cent sweet pea diet for 8 weeks. Note that the deeply stained hyalinizing fibrosis is prone to undergo calcification. There is uneven basophilia in the hyaline area with fused corpuscles of incipient calcification. This represents a relatively mild degree of dystrophic calcification of the hyalinizing fibrosis that may be found in lathyrism. (Hematoxylin and eosin stain; orig. mag. X750.)

DISCUSSION

Injury to the Periodontal Ligament Resulting From the Lathyrus Toxin. Damage to the periodontal ligament by lateral compression has been recognized repeatedly and has been described in detail by Sandstedt,[13] Oppenheim,[14] Orban[15] and Reitan.[16] However, few investigators have recognized similar injury due to systemic toxins present in legumes. Interference with the blood supply produces hyalinization of the periodontal ligament and is a consequence of the lathyrus toxin. In lathyrism an alteration occurs in the pattern of attrition, so that the ligament is damaged, in part, because the masticatory forces of attrition move the tooth beyond the range of physiologic positions.

Lathyrism is one of the very few known diseases that produces such visible and widespread lesions in the periodontal ligament. This fact may be due to the dual nature of the lathyritic influence placed upon the ligament, namely abnormal attritional forces plus the localized action of the lathyrus toxin directly upon the ligament. It has been hypothesized and it appears almost certain that the single principal fiber does not extend from bone to cementum, but that much shorter fibers are united together to form the periodontal principal collagenous fibers. If the hypothesis is correct, then injury to the periodontal ligament should produce a break in the single fibers at the areas of union rather than produce a tear of the whole fiber strand. In lathyrism, the alteration occurs principally in the center of the periodontal ligament with a consequent loosening of the teeth without a tear of the whole fiber. It is possible that the union of the single fiber occurs in the region of hyalinization which develops in the approximate center of the ligament.

The very few reports in the literature concerning direct influence upon this ligament as an exceptional behavior, indicate that alveolar bone is the initial tissue in-

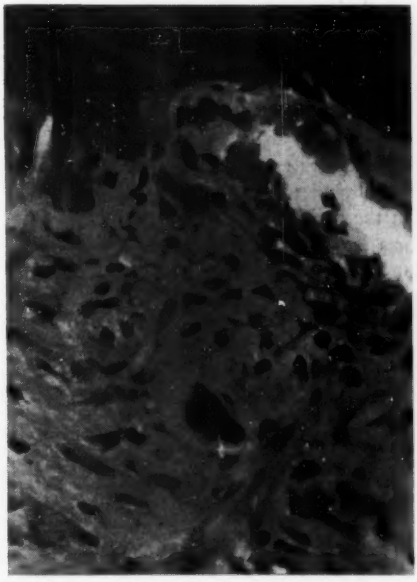

Fig. 5A. Photomicrograph of the periodontal ligament from a male rat fed a 30 per cent sweet pea diet for 8 weeks. Note the densely stained calcified mass within a relatively small eosinophilic deposit in the pathognomonic lesion of lathryism. (Hematoxylin and eosin stain; orig. mag. X800.)

Fig. 5B. Photomicrograph of the periodontal ligament from a male rat fed a 30 per cent sweet pea diet for 8 weeks. Note the uneven basophilia and more diffuse calcification in a small spherical area of the amorphous hyaline or eosinophilic material. (Hematoxylin and eosin; orig. mag. X750.)

volved because it receives the blood supply first. If experimental lathyrism results in direct damage to the periodontal ligament it may similarly explain the observations reported in this ligament. An injury of the kind involved in lathyrism would eliminate or at least diminish the high resistance of a normal periodontal ligament and thus open the way for excessive occlusal forces to involve the ligament very readily. The lathyrus toxin sensitizes the ligament to any small alteration in the forces of occlusion and attrition.

Absence of Inflammation in the Periodontal Ligament During Experimental Lathyrism. The findings in the present investigation suggest that trauma from altered attrition may be followed by some type of attempt (pathologic) at regeneration. The periodontal ligament which was decidedly damaged during experimental lathyrism, was entirely free of inflammatory cells, and showed a response that may be considered an attempt at regeneration. Since traumatism plays a part in the changes found during lathyrism, one may conclude that trauma by itself, or in combination with exogenous toxic agents does not cause inflammatory changes in the periodontal ligament.

Molar Periodontal Ligament Versus Incisor Ligament. In examining the differences in reaction between the incisor and molar periodontal ligaments to the lathyrus toxin one is immediately confronted with the fact that lathyrism produces in the molar ligaments more severe alterations, not only in the dental tissues themselves but also in the relationship between the tooth and the alveolar bone. This is due to the fact that the incisors of the rat are teeth of continuous growth.

Teeth with limited growth, i.e., molars show a quantitative, as well as a principal difference in the structure of the periodontal ligament. It is extremely difficult to

trace a single principal fiber from cementum to bone. The recognition of a plexus intermedius in the molar periodontal ligament is of great significance for the alterations that occur in this zone are incapable of normal recovery during lathyrism due to the limited and final cessation of growth in this ligament. However, supplementation therapy[17,18,19] with proteins and amino acids was found effective in the periodontal ligaments of both molars and incisors. Histologic alterations in the incisor periodontal ligament could not be recognized after dietary supplementation. In the molar ligament supplementation was partially effective since histologic changes showed a decrease in the number and size of lathyritic bodies. In the specialized mesenchymal structure supporting the teeth (periodontal ligament), the initial migration and proliferation of immature fibroblasts resembles the early phase of fibrotic repair. Such a concentration of immature cells at the sites of injury to the principal fibers is predictable on the basis of the law that responses to injury always involves the concentration of reserve, mobile, and replaceable cells at the site of injury. The sites of injury are focal and numerous in the periodontal ligament corresponding to areas of intertwining of fibers to form the continuous principal fibers. This produces a response that appears at the onset as diffuse or certainly more widespread. The adult cells remaining in the adjacent periodontal ligament undergo differentiation, expand and migrate along the lines of pull of the principal fibers. This atavism on the part of differentiated cells of the periodontal ligament has not been reported previously. Therefore, the migration of young fibroblasts of the periodontal ligament into a palisade arrangement which proliferate and undergo a metaplastic change is a unique, specific, and conspicuous alteration in lathyrism. Here the stimulus to fibroblastic proliferation is apparently not due primarily to loss of adult functioning fibroblasts but instead to the lathyrus toxin itself.

As rapidly as two weeks after initiating the lathyrus diet, the middle zone of the periodontal ligament shows differentiation of fibroblasts and the adjacent connective tissue underwent some irregularities with formation of eosinophilic homogeneous bodies. This was due to the laying down of excessive zones or bands of dense collagen bundles by these cells. Regeneration in the periodontal ligament during lathyrism is similar to tendon regeneration inasmuch as the specialized structures present in the periodontal ligament appears to evolve from indifferent connective tissue following injury, with abnormal fibrosis, which results after cell migration to sites of intertwining of the smaller fibers.

When the stromal lines of tension are interrupted by the action of the sweet pea toxin injury, edema and hydration of stromal matrix induces a proliferation of mesenchymal elements. The reaction of repair in lathyrism proceeds rapidly in the

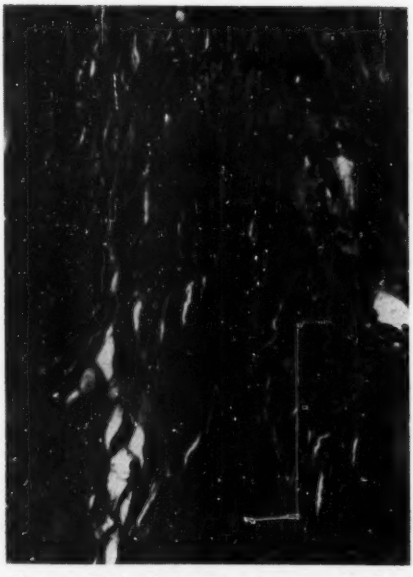

Fig. 6. Photomicrograph of the periodontal ligament from a male rat fed a 50 per cent sweet pea diet for 3 weeks. Note the almost fluid or transudative appearance of the noncellular hyaline lesion of lathyrism, which was observed in this study to stain deeply blue, showing the staining reactions of collagen. The internal fibrillar structure is not visible with this stain. (Masson's stain; orig. mag. X600.)

periodontal ligament. It is no doubt true that the accumulation of metaplastic cells in the periodontal ligament during lathyrism is the result of a reactive proliferation following chemical, toxic and mechanical injury and possibly death to other members of the tissue of cells. This replacement of destroyed cells by a proliferation of metaplastic cells, is not a direct result of the lathyritic toxin but a pseudoform of repair.

The exact mechanism by which beta-aminopropionitrile brings about this effect during experimental lathyrism is not at the present time well understood. However, once the beta-aminopropionitrile causes the alteration in the periodontal ligament the morphology of the fibroblasts that proliferated in the ligament of experimental animals is different from that of the fibroblasts in the ligament of the controls. Furthermore, the response in the experimental ligaments differs slightly in different parts of the ligament being more severe in the apical one half. Firstly, in the periodontal tissue, the proliferation and differentiation of the fibroblast in response to the stimulus of the sweet pea is enhanced compared to that of the controls. Secondly, the apical region of the ligament is an area where considerable mechanical stress can be assumed to be present, the fibroblast responds by a more intense proliferation and has a tendency to be more metaplastic than in the controls. It is, however, difficult to explain exactly why the morphology of the fibroblastic response in the experimental rats differs slightly in different parts of the periodontal ligament. The connective tissue fibers may be a form of abnormal collagen[12] that develops in areas of stress.

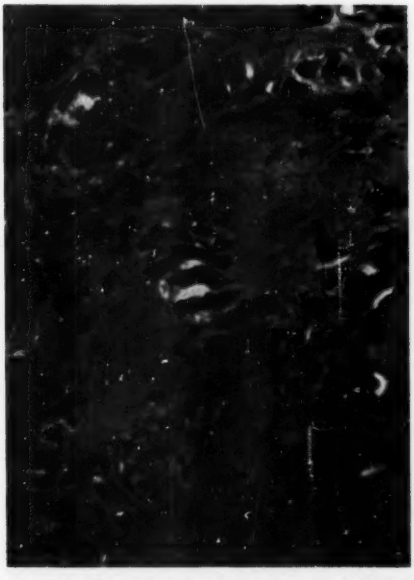

Fig. 7. Photomicrograph of the periodontal ligament from a male rat fed a 50 per cent sweet pea diet for 2 weeks. Note the homogeneous amorphous material adjacent to the surface of bone. The two areas are separated by hyperchromatic fibroblasts. Such deposits are not metachromatic with crystal violet. With Masson's stain it gives a blue reaction consistent with collagen. (Masson's stain; orig. mag. X800.)

Stimulation to Calcification in Limited Areas of the Periodontal Ligament. The beta-aminopropionitrile content of the sweet pea plays the role of an inductor to calcification during experimental lathyrism.

During lathyrism one is definitely able to distinguish between inherent osteogentic potency and calcification by induction.

Thus it seems reasonable to assume that inherent osteogentic potency is not the factor in lathyrism but calcification occurs by induction.

The nature of the principle which initiates and sustains the presence of calcification is one of the most important problems of osteogenesis.

In the rat calcification is not produced simply by injuring the periodontal ligament by either mechanical or chemical influences.

In lathyrism one recognizes the role of systemic factors capable of influencing the periodontal tissues relatively far removed from the gingival crevice exhibiting bodies or lakes of eosinophilic material. This is one of the very few instances in which alterations occur in the periodontal liga-

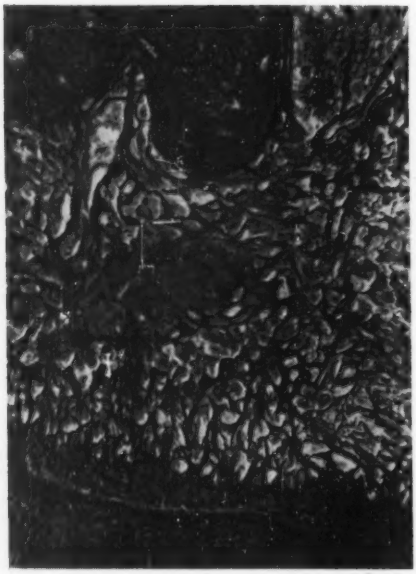

Fig. 8. Photomicrograph of the periodontal ligament from a male rat fed a 50 per cent sweet pea diet for 3 weeks. Note the masking of the non-cellular reticulum due to a thickening or swelling of the reticulum in a lesion of lathyrism. Note the appearance of a fluid or transudate where the masked fibers meet to form this material. It stained from grey to black in its reaction with Wilder's stain. (Wilder's reticulum stain; orig. mag. X600.)

ment proper without any local contributory involvement of the gingival or crevicular epithelium. The only other reported situation in which this occurs is during the early stages of periodontosis.

SUMMARY AND CONCLUSION

It is concluded that modification of lathyritic alterations (lathyritic bodies) were significantly influenced through a change in the proportion of dietary proteins and amino acids. Within limits that permit relatively good growth and body weight, we have unequivocally demonstrated a reduction in lathyritic changes, particularly lathyritic bodies, of the periodontal ligament. The influence of dietary proteins and amino acids has been reported to be reflected in the liver, but this investigation reflects its influence in extrahepatic tissues, as the periodontal ligament. Probably the observed effects of lathyritic bodies are related to amounts of protein in the periodontal tissues that are marginal with respect to optimal periodontal tissue health and function of this supporting structure. Perhaps the establishment of similar critical conditions in other tissues, with carefully controlled dietary intake and body weight, might also reveal the influence of protein and amino supplementation on the intimate changes leading to the formation of alterations at these sites. The increased interest and work of the past decade has resulted in a broadening recognition of the relationship of nutrition to disturbances of connective tissue metabolism. This experiment provides, perhaps only generally and crudely, the significance of the proportion of dietary protein and amino acids in the genesis and growth of disturbances in connective tissue. One cannot predict beforehand the importance of these isolated facts and correlations that have been presented here. In the future they may be integrated with information from other fields to furnish practical working tools.

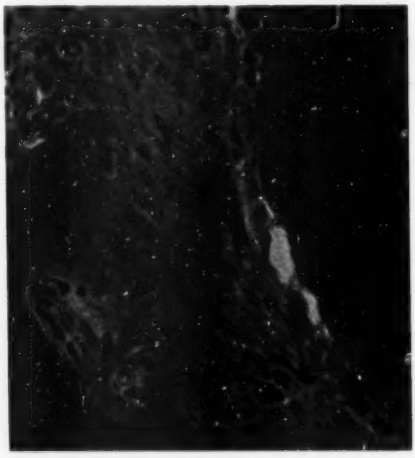

Fig. 9. Photomicrograph of the periodontal ligament from a male rat fed a 30 per cent sweet pea diet for 8 weeks. Note the large and homogeneous appearance of this material, which was observed in this study to vary from orange red to deep red in its reaction with van Gieson's stain. Such deposits are not metachromatic with crystal violet or toluidine blue. (van Gieson's stain; orig. mag. X800.)

The following experimental findings were present in the molar periodontal ligament of protective diet I and S.P. III animals:

(1) Prominent palisading with advanced and numerous lathyritic bodies,

(2) Absence of ankylosis,

(3) Marked reduction in vascularity,

(4) Absence of epithelial remnants,

(5) Decreased width of periodontal ligament,

(6) Absence of distal drift of molars,

(7) Absence of inflammation,

(8) Absence of dystrophic calcification in lathyritic bodies in P.D. I; presence of dystrophic calcification in S.P. III animals, and

(9) Disruption of the majority of principal fibers of the periodontal ligament.

The above findings were either absent or greatly diminished in severity in animals fed of P.D. II to VII groups.

The following experimental findings were present in the incisor periodontal ligament of protective diet I and sweet pea diet III animals:

Fig. 10. Photomicrograph of the periodontal ligament of a male rat fed a 30 per cent sweet pea diet for 8 weeks. Note the large area of hyaline material. This was strongly eosinophilic, red with van Gieson's stain, and non-metachromatic with toluidine blue. The material gave a very weak positive reaction with periodic acid-Schiff stain. (Periodic acid-Schiff stain; orig. mag. X800.)

Fig. 11. Photomicrograph of the periodontal ligament of a male rat fed a 30 per cent sweet pea diet for 8 weeks. Note the incipient lakes of eosinophilic material developing in the middle of the ligament in a focus of normal tissue. The eosinophilic material is present only between the cells in the heavily stained areas. (Hematoxylin and eosin stain; orig. mag. X800.)

(1) Normal width of periodontal ligament,

(2) Absence of ankylosis,

(3) Normal vascularity,

(4) Presence of epithelial remnants,

(5) Dilatation and hyperemia of blood vessels,

(6) Increased thickness and dystrophic calcification in labial alveolar periosteum in P.D. I animals; normal labial alveolar periosteum width in S.P. III animals,

(7) Lathyritic bodies absent in cemental fibers of periodontal ligament,

(8) Edema and disorientation of cells in plexus intermedius,

(9) Irregular homogeneous lakes in alveolar fibers of P.D. I; absent in S.P. III animals, and

(10) Absence of inflammation in periodontal ligament.

The above alterations were absent in P.D. II to VII, S.P. I and II groups.

This investigation provides indications concerning the possible influence of known and relatively well-defined components of diets. However, natural foods contain a number of constituents which have been given little attention in nutrition as well as in cancer research because they apparently are not dietary essentials. In addition, there must be others yet undetected. Perhaps among these unregarded substances are some with toxic or carcinogenic activity; and others that potentiate or oppose the action of toxins and carcinogens. There is a danger that findings may be added to suggestions and guesses to build up concepts which by repetition become accepted. This is not the purpose of this study, since we have respect for facts and recognize the pitfalls of speculation.

The results of this investigation readily provides indications that a search for carcinogenic and toxic compounds in human dietary regimens might well be worthwhile. Early nutrition research had to be content with fact finding, and this has been accomplished to a satisfactory degree. More and more attention is being turned to studying the mode of action of those diet alterations which affect the genesis of toxic or carcinogenic alterations. This work may proceed to a better understanding of toxinogenesis itself.

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Editorials . . . .

The Old Changeth . . . Or Does It?

On August 3, 1859, 26 dentists met in Niagara Falls, New York, to form the first national dental organization which they ambitiously named "The American Dental Association". At that time dentistry was playing a very minor role in the field of health care; the importance of oral health was not appreciated and dental services were not in great demand. Dental-treatment procedures were crude, inadequate, often painful, and the understanding of dental diseases was scanty.

The past century has seen a remarkable growth in dentistry from almost every standpoint. During this time the dental profession has grown in competency, in numbers, in stature, and is now a well-respected member of the health team. Development in all phases of dentistry—anesthetics, precision-treatment techniques, dental equipment—and a better understanding of oral ills has resulted in an improvement of the effectiveness of dental therapy and in the acceptance by the public of dental services. Remembering the lowly status of dentistry one hundred years ago, certainly "the old order changeth". The American Dental Association has been one important element in the upgrading of dentistry and so on its one hundredth birthday, deserves commendation.

Improvement in the status of dentistry came about because of the high principles and dedicated services of a host of dentists working alone and through organizations. Continued progress of dentistry will be dependent upon the contributions of unselfish, devoted leaders. Certainly the dental profession still has many members who are concerned with the progress of dentistry, but there is also a growing number whose interest in methods of justifying high fees for dental service comes ahead of their interest in improving the quality of dental treatment. During the next century the American Dental Association must continue to exert its influence first toward improving the oral health of the public, and secondarily, toward improving the status of the dental profession. This will require future leaders whose integrity, unselfishness, and idealism equal the best of the past. We hope in this regard that the old order changeth not.

Abraham Lincoln's Teeth

Since this is the one-hundred and fifteenth anniversary of Abraham Lincoln's birth, it was inevitable that someone ask "What do you know about Lincoln's teeth?" The editor's answer was, "Very little," and a survey of available references suggests little is known.

Apparently Lincoln was blessed with "beautiful, white teeth," though he did have at least three extractions. He purchased a toothbrush in 1843, but no account of its use could be found. We would appreciate additional information on the status of Lincoln's dentition.

Announcements . . . .

PROGRAM FOR ANNUAL MEETING AMERICAN ACADEMY OF PERIODONTOLOGY

SEPTEMBER 10, 11, 12, 1959
Barbizon Plaza Hotel, New York, New York

Many committees are responsible for planning and presenting the annual meeting of the American Academy of Periodontology. Dr. Clarke Chamberlain, the efficient secretary and his able assistant, Mrs. Lorrie Hill, of course are responsible for many of the important details associated with the meeting. Also the President, this year Dr. Joseph L. Bernier, works diligently in a general supervisory capacity to assist on all committees. Dr. James E. Aiguier, the Treasurer, also deserves credit and helps to make the meeting run smoothly.

The 1959 scientific sessions are under the supervision of Dr. B. O. A. Thomas, assisted by Drs. Nachazel and Ramfjord. The program has been planned by Dr. Irving Glickman with Drs. Gold, Neilson, Ratcliff and Weiner on his committee. Clinics have been arranged by Dr. Frank Beube, Chairman, and Drs. Chaikin, Chasens, Hileman and Paulsen. The Local Arrangements Committee consists of Dr. Craft Hopper, Chairman, and Drs. Cohne, Gottsegen, Reeves and Stahl.

The Round Table Luncheon has been planned by Dr. Justin Towner and Dr. Harold J. Leonard. Registration is under the direction of Dr. Clarence Schmidt assisted by Drs. Fraleigh, Paul, Robinson, Stoker, Swenson, Swindle, and Wiebusch. Entertainment has been planned by Dr. Leonard Hirschfeld, assisted by Drs. Brayshaw and Kramer.

Properties have been arranged for by Dr. George Witkin, Chairman and Drs. Halik, Morris and Older, and of course as usual publicity under the capable direction of Dr. Howard Hartman assisted by Drs. Eddie L. Ball and Francis J. Fabrizio.

THURSDAY, SEPTEMBER 10, 1959

11:00 a.m.—*Session Chairman*—J. W. Neilson, B.A., D.D.S., M.S.
Dean of Dentistry, The University of Manitoba, Winnipeg, Canada.

New Approaches to the Study of Mucous Membrane Disease, Herbert Mescon, M.D.
Professor and Head, Department of Dermatology, Boston University School of Medicine and Massachusetts Memorial Hospitals, Boston, Massachusetts.

11:45 a.m.—Discussion

2:00 - 5:00 p.m.—Symposium—Desquamative, Bullous, Ulcerative and Other Lesions of the Oral Mucosa—Their Etiology, Clinical and Microscopic Features and Treatment.

Moderator—Harry Lyons, D.D.S., D.Sc.
Dean Medical College of Virginia, School of Dentistry, Richmond, Virginia.

Participants—J. J. Pindborg, D.D.S., Dr. Odont.
Danmarks Tandlaegehojskole Royal Dental College, Copenhagen, Denmark.
"Etiology and Clinical Features"

Lester R. Cahn, D.D.S., F.D.S.R.C.S. (Eng.) F.D.S.R.C.S. (Edin.)
Associate Professor of Oral Pathology, Columbia University, Attending Pathologist the Mt. Sinai Hospital, New York, New York
"Histopathologic Analysis"

Hamilton B. G. Robinson, D.D.S., M.S.
Dean, University of Kansas City Dental School, Kansas City, Missouri
"Treatment"

FRIDAY, SEPTEMBER 11, 1959

9:00 a.m.—*Chairman*—Sidney Sorrin, D.D.S.
Professor and Chairman of the Department of Periodontia and Oral Medicine, New York University, College of Dentistry, New York, New York

Constructive Critique of the Current Status of the Treatment of Periodontal Disease

Harold G. Ray, D.D.S., M.S., Professor of Periodontology, University of California School of Dentistry, San Francisco, California

9:35 a.m.—Discussion

10:00 a.m. - 12 noon—Television Presentation from the Armed Forces Institute of Pathology, Walter Reed Hospital, Washington, D. C.

"Beyond the Periodontium"
Laboratory procedures and medical consultation will be utilized to explore the systemic condition of patients with periodontal problems.

2:00 - 5:00 p.m.—Symposium—Pathways of Periodontal Progress—Some Representative International Contributions in the Past Quarter Century.

Moderator—Maynard K. Hine, D.D.S.
Dean Indiana University School of Dentistry, Indianapolis, Indiana

Participants—Hans R. Mühlemann, M.D., D.D.S.
Professor of Operative Dentistry and Periodontology, University of Zurich Dental Institute, Zurich, Switzerland
"10 Years of Tooth-Mobility Measurements"

A. J. Held, Professor of Endodontia and Paradonta, Institut Universitaire de Medecine Dentaire, Geneva, Switzerland
"The Systemic Approach to the Treatment of Periodontal Disease"

Fermin A. Carranza, Sr., D.D.S., Professor of Periodontia, Faculty of Dentistry, University of Buenos Aires, Buenos Aires, Argentina
"The Problem of Reattachment in Periodontal Therapy"

W. G. Gross, M.B., M.S., B.D.S., M.R.C.S., L.R.C.P., L.D.S., Senior Lecturer and Head of Department of Periodontology, Institute of Dental Surgery,

Eastman Dental Hospital, London, England
"The Use of Bone Implants in the Treatment of Infrabony Pocket"

Jens Waerhaug, DD.S., Ph.D., Professor of Dentistry and Head of the Department of Periodontia and Crown and Bridge, Norwegian State Dental School, Oslo, Norway
"Restorations as Etiologic Factors in Periodontal Disease"

SATURDAY, SEPTEMBER 12, 1959

9:00 - 11:30 a.m.—Symposium—The Bone Destructive Pattern in Periodontal Disease.

Moderator—B. O. A. Thomas, B.A., M.S., D.D.S., Ph.D.
Clinical Associate Professor, College of Physicians and Surgeons, San Francisco, California

Participants—Lent C. Johnson, M.D.
Chief of Orthopedic Pathology, Armed Forces Institute of Pathology, Washington, D. C.
"The Mechanisms of Bone Formation and Bone Resorption and the Factors Which Affect Them"

Sigurd Ramfjord, L.D.S., M.S., Ph.D., Professor of Dentistry (Periodontics), University of Michigan School of Dentistry, Ann Arbor, Michigan
"Pattern of Bone Destruction in Periodontal Disease Caused by Local Factors"

William G. McIntosh, D.D.S., M.Sc.D., Associate Professor of Periodontology, University of Toronto School of Dentistry, Toronto, Ontario, Canada
"The Clinical Management of Varied Bone Destructive Patterns in Periodontal Disease"

RESEARCH FORUM

2:00 - 5:00 P.M., SATURDAY, SEPTEMBER 12, 1959

2:00 - 2:15—Discussion—"A Study of the Effect of the Drug BO-175 on the Rate of Bone Regeneration in the Tibia of Guinea Pigs"
Dr. Richard Stallard, University of Minnesota

2:20 - 2:40—Discussion—"Some Observations on Bone Resorption in Tissue Culture"
Dr. Paul Goldhaber, Harvard School of Dental Medicine

2:45 - 3:05—Discussion—"The Muco-polysacharides of the Gingiva and their Relation to Periodontal Disease"
Dr. John C. Thonard, University of Pittsburgh

3:10 - 3:25—"Studies on the Antibacterial Action of Human Saliva"
Dr. Bernard J. Zeldow, University of Washington

3:25 - 3:45—Discussion of Previous papers and Future Periodontal Research
Dr. Howard Myers, University of California

3:45 - 3:55—Discussion—"Glomi as Vascular Components in the Periodontal Membrane"
Dr. D. Vincent Provenza, Baltimore College of Dental Surgery, University of Maryland

4:00 - 4:15—Discussion—"Factors Governing the Deposition of Cementum in Surgically Created Pockets"
Dr. Melvin L. Morris, Columbia University

4:20 - 4:35—Discussion—"Experimental Periodontal Lesions Induced by Utrasonics"
Dr. George W. Burke, Medical College of Virginia

4:40 - 4:55—Discussion—"The Prevalence Morphology and Distribution of Epithelial Rests in the Periodontal Ligament of Humans"
Dr. Charles M. Reeve, Loyola University, Chicago

PROJECTED CLINIC PROGRAM

"Integration of Periodontal Tissue Therapy to Restorative Dentistry"

1:30 p.m.—"Occlusal Aspects of Periodontics"
Ernest Baker, D.D.S., Oakland, California

1:50 p.m.—"Relationship of the Gingival Margin of the Restoration to the Sulcus"
D. Walter Cohen, D.D.S., University of Pennsylvania, Philadelphia 4, Pa.

2:10 p.m.—"Restorative Dentistry as an Adjunct of Periodontal Therapy—A Case Report"
Captain H. H. Fridley, D.D.S., M.S.D., U. S. Naval Dental School, Bethesda 15, D. C.

2:30 p.m.—"Periodontal Tissue Therapy in Relation to Proper Performance of Restorative Dentistry"
Gerald M. Kramer, D.M.D., 9 Nahant Street, Lynn, Massachusetts

2:50 p.m.—"The Effects of Various Types of Restorations on Periodontal Health"
John J. Lucca, A.B., D.D.S., Columbia University, School of Dental and Oral Surgery, 630 West 168th Street, New York 32, N. Y.

3:10 p.m.—"The Conflict Between Crown and Bridge Restorations and The Interproximal Tissues and the Gingival Sulcus"
Julius W. Obin, B.A., D.D.S., 366 Madison Ave., New York 17, N. Y.

3:30 - 4:00 p.m.—General Discussion

UNIVERSITY OF WASHINGTON

The University of Washington School of Dentistry is now offering a twenty-four months course in ORAL PATHOLOGY leading to the degree of Master of Science in Dentistry (M.S.D.) beginning in September, 1960. The course will be under the direction of Leo M. Sreebny, A.B., D.D.S., M.S., Ph.D. Special emphasis will be given to training in Research in addition to the offering of a sound background in clinical Oral Pathology. The submission of a satisfactory thesis reporting on research performed will be a partial requirement. More definite information is available in the Graduate School Bulletin.

All communications should be addressed to Saul Schluger, D.D.S., Director, Graduate Dental Education, University of Washington School of Dentistry, Seattle 5, Washington.

THE ROCKY MOUNTAIN DENTAL SEMINAR

The Rocky Mountain Dental Seminar, sponsored by the Columbine Periodontal Study Club of Denver, Colorado and the Michael Reese Histopathology Study Club of Chicago, Illinois will be held in Glenwood Springs, Colorado, December 13-19, 1959. The subject will be Advanced Concepts in Periodontics.

This Seminar will honor Dr. Harry Sicher who will also be a member of the panel. Others on the panel are Saul Schluger, Seattle, Washington and Balint Orban, Denver, Colorado. The Moderator will be Dr. Saul Levy of Chicago, Illinois.

The clinicians of the Seminar are: Arnold A. Ariaudo, San Diego, California; Nathan Friedman, Beverly Hills, Calif.; Daniel A. Grant, San Diego, California; Harold E. Grupe, Amarillo, Texas; William H. Hiatt, Denver, Colorado; Gerald P. Ivancie, Denver, Colorado; Claude L. Nabers, San Antonio, Texas; Cliff Ochsenbein, Austin, Texas; John Prichard, Fort Worth, Texas; Nicholas Marfino, Buffalo, New York; Harry Staffelino, Chicago, Illinois; M. N. Wilderman, Chicago, Ill.

Further information may be obtained by writing the Seminar secretary, Mrs. James Manella, 1700 Mesa Road, Colorado Springs, Colorado.

MASSACHUSETTS MEMORIAL HOSPITALS

The Department of Stomatology, Division of Graduate Studies School of Medicine, Boston University and the Massachusetts Memorial Hospitals announce the following postgraduate courses:

Periodontal Therapy: October 19-24, 1959, Dr. Henry M. Goldman, Dr. Bernard S. Chaikin and Staff

Occlusal Adjustment: November 2-6, 1959, Dr. Henry M. Goldman, Dr. Bernard S. Chaikin and Staff

Oral Pathology: January 11-16, 1960, Dr. Jack Bloom and Dr. Henry M. Goldman

For further information and application write to Director, Post Graduate Courses, Department of Stomatology, Massachusetts Memorial Hospitals, 750 Harrison Ave., Boston 18, Mass.

UNIVERSITY OF PENNSYLVANIA

A course on DENTISTRY FOR HANDICAPPED CHILDREN will be presented by Dr. Manuel M. Album and Associates 30th November; 1st and 2nd December 1959. It is arranged to give the practitioner a better understanding of the dental problems and treatment of children who suffer from cerebral palsy, mental retardation, muscular dystrophy, multiple sclerosis, and other handicapping conditions.

A three day course will be presented on CEPHALOMETRY by Dr. Viken Sassouni on 23, 24, 25 November 1959. This course is designed to prepare the clinician for the use of cephalometry for diagnosis, prognosis, treatment progress evaluation and growth appraisal. Special emphasis is put in this course on the practical applications of cephalometry for everyday use.

A one day course on THE USE OF ULTRASONICS IN PERIODONTIA will be presented by Drs. Henry M. Goldman and D. Walter Cohen 12th December 1959. This course will encompass the principles of ultrasonics and the tissue reaction to the use of these instruments.

For further information, please write to Postgraduate Courses, School of Dentistry, University of Pennsylvania, 4001 Spruce Street, Philadelphia 4, Pennsylvania.

UNIVERSITY OF MINNESOTA

The Division of Oral Pathology of the School of Dentistry, University of Minnesota, is offering a Fellowship in Clinical Oral Pathology sponsored by the American Cancer Society for the period 1 July 1960—30 June 1961. This will pay the recipient $3,600.00 per annum tax-free and is renewable upon approval of the ACS.

A program of education has been synthesized to give the trainee advanced experience in both clinical and basic material in dermatology, dermatopathology, biostatistics, tumor pathology, advanced oral pathology, pediatrics, and metabolic and endocrine disorders. A wealth of clinical material is available within the University of Minnesota Medical Center.

Applicants must apply before 15 February 1960, forwarding a curriculum vitae and academic record. Inquiries should be addressed to: Dr. Robert J. Gorlin, Chairman, Oral Pathology, School of Dentistry, University of Minnesota, Minneapolis 14, Minnesota.

SOCIETY OF ORAL PHYSIOLOGY AND OCCLUSION

The sixth annual meeting of the Society of Oral Physiology and Occlusion will take place on Saturday, December 5, 1959 in the Empire Room at the Hotel New Yorker, 34th St. and 8th Ave., New York City. The seminar will be scheduled from 9 A.M. to 5 P.M. and the subject will be "Pain and the Temporomandibular Joint." Among the speakers will be Dr. Janet Travell, Dr. Joseph J. Martini and Dr. Clyde B. Kyne. Those interested in joining this organization should get in touch with Dr. S. M. Kelner, Sec'y, 57 West 57th St., New York City.

Letters to the Editor . . . .

Dear Sir:

I was interested to read the article by Drs. Lloyd and Baer entitled "A Permanent Fixed Amalgam Splint" published in the April Number of the Journal of Periodontology which I have just received.

I described a splint on these lines in Parodontologie in 1954, No. 3, Page 119. I used a method of forming the matrix which was almost exactly similar; the matrix was not, however, hinged distally but in two separate parts held together by wires or very small bolts passing through the acrylic in the interdental spaces. Several types of strengthener were used including stainless steel wire of various sections and also twisted stainless steel wire. The matrix was left in position for twenty-four hours so as to prevent undue strain on the interdental portions of amalgam during the setting period. The acrylic was then removed and final trimming and polishing of the amalgam restorations carried out.

In the rather limited number of cases undertaken, however, it was found, after variable periods of time, that several of these cases exhibited fractures of the amalgam, usually close to the embrasure, and although one or two cases have still got successful amalgam splints of this type after five years, there was failure in the majority of cases and the method was reluctantly discontinued.

W. G. Cross, M.B., M.S., B.D.S.,
Eastman Dental Hospital,
Gray's Inn Road, London, W.C.I.

Dear Sir:

Dr. Cross was kind enough to send us a reprint of his article, "The Importance of Immobilisation in Periodontology", which appeared in, Parondontologie, No. 3, page 119, 1954. Although Dr. Cross goes into detail about the construction of a matrix in his letter, no mention is made in his article about the use of, necessity for, or construction of such a matrix. Without a plastic matrix to hold the mobile teeth rigid during condensation of the amalgam, successful amalgam splints cannot be made.

Our oldest splint to date, is two years old. In the limited number of cases where it has been used, no fracture of the enamel has occurred at any time. Occasionally, where the preparation was too shallow, a fracture did occur in the amalgam close to the embrasure. It has not been necessary to remake the entire splint when this happens however, as a simple repair can be made in the amalgam at the area of fracture.

As this splint is used only in advanced cases of periodontal disease, the margins of the fillings are all well above the gingival tissues. The tissue reaction around our splints, therefore, is good, where home care is good.

Preliminary studies using metal implants within the amalgam splint, indicates that although they may prove to be helpful in preventing fractures, the most important factors are probably a careful check of the occlusion, and preparations of adequate depth.

Dr. Ralph S. Lloyd, Chief
Dental Department, Clinical Center
Bethesda 14, Md.